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COLLECTION AGENCIES

Veterinarians continue to report unsatisfactory dealings with so-called collection agencies, so we consider it advisable to warn our members again to be on their guard against entering into contracts with concerns that they know nothing about. In the first place, the American Veterinary Medical Association has not approved or endorsed any collection agency, either directly or indirectly, and probably will refrain from doing so in the future. If an agent of one of these concerns makes such a representation, just ask him to show proof of his claim. It would appear that a favorite practice of agents soliciting business for some of these agencies is to make representations that are not embodied in any printed contract that the unsuspecting veterinarian is asked to sign on the dotted line. If you sign a contract, read it carefully first, and see that you get an exact copy of the contract which you sign. Several complaints have been filed recently, by veterinarians who believed that the collection agencies had not lived up to their part of the agreement, and when we asked for copies of whatever it was that they had signed, to see if there had been any breach, the veterinarians were obliged to admit that they did not have copies of the contracts. Various methods are used by the concerns that make a business

of collecting bad accounts. The writing of threatening letters to debtors seems to be a favorite practice of some. The same concern may have more than one method and more than one form of contract. Too often the "minimum fee" clause not only leaves nothing for the veterinarian on collections actually made, but he finds himself called upon to pay a bill for services rendered by the agency. We hope that this word to the wise will be sufficient.

CASE REPORTS

Why do our veterinary journals contain so few case reports? Undoubtedly there are a number of reasons. Or should we say "excuses"? The first question has been put to approximately a dozen veterinarians, most of whom are practitioners, during the past six months. It has been interesting to receive and analyze the answers to the question.

Procrastination appears to play a prominent part, as several veterinarians readily admitted that they had been asked and had intended to write up reports of certain interesting cases but had "just put it off," for no good reason at all. Inability to write was given as a reason by some. This should not be taken literally, however. What they meant was that they had not had very much experience in writing. This never need be an excuse. Just send in the essential facts and we will put them in shape for publication.

One veterinarian, who evidently had more than his share of modesty, replied to the effect that he was not looking for publicity. His answer stumped us for the moment. A further exchange of confidences seemed to remove any ideas that he may have entertained concerning breaches of professional ethics and we parted after he had promised to write up an interesting case or two. This was over four months ago. We are still waiting for the reports.

"Lack of time" appeared rather frequently in our tabulation of replies. It is true that in most instances those who are best able to supply the kind of material under discussion are busy practitioners with years of experience behind them. This experience is of great help in selecting the cases to report and in evaluating methods of treatment and operative procedures. Therefore, among our experienced practitioners, theoretically, at least, should be found the most productive field for securing case reports. But the results are hardly in keeping with the theory expressed.

One practitioner whom we interviewed stated that he would like to see more case reports published in the JOURNAL each

month. He admitted that the "scientific" material had to be published "to advance the prestige of veterinary science," but we ought to have more case reports. Then we got up enough nerve to ask him how many he had contributed during the past ten years. He was obliged to admit—with more than just the suggestion of a blush—that he had not been able to "get around to writing any."

Another veterinarian was inclined to place the blame on our educational institutions. He said that he had received absolutely no instruction in keeping case records, let alone writing up case reports. This man had been out of college for almost twenty years and we had no reason to question his statement. The fact remains, however, that an important part of the curriculum of any veterinary college is proper instruction in keeping case records, in evaluating methods of treatment and in analyzing results of treatment in a series of similar cases. If a veterinarian is familiar with the *modus operandi* of case recording, it should be possible for him to derive much genuine pleasure from studying his records. Valuable information, from the standpoints of both diagnosis and treatment, is sometimes disclosed by a critical study of a series of cases.

What was said in the preceding paragraph brings out another point on which there has been some confusion. A case report need not consist of a mere recording of the birth of a freak calf or the finding of an unusually long worm. These have their interest but contribute very little to the advancement of our knowledge of veterinary medicine.

TO THE RESCUE

At the suggestion and with the help of Dr. J. V. Lacroix, editor of the *North American Veterinarian*, and with the full support of Dr. D. M. Campbell, editor of *Veterinary Medicine*, the editor of the JOURNAL of the A. V. M. A. has started the machinery in motion for raising and administering a fund to be used for assisting veterinarians in the flood-stricken areas. The first move was to get out a letter to veterinarians in all parts of the United States. The following message was sent to more than 7,000 members of the profession, under date of January 27:

Fellow Veterinarian:

We appeal to you for assistance . . . to contribute to a fund to help members of our profession who are in distress.

This S. O. S. is being addressed to every veterinarian in the United States outside of the flood areas. It is not intended to so-

licit those who are within the water-devastated territory.

The purpose of this appeal is to solicit your contribution to a fund that will be used for aiding veterinarians who have suffered serious losses as a result of the flood.

This fund will be administered by the appointed representatives of the American Veterinary Medical Association, as promptly and as equitably as conditions will permit, to aid deserving veterinarians who are in distress. An accounting will be made of all moneys received and expended.

As we write this message, the toll in loss of life, disease, suffering and destruction of property in the flooded areas is on the increase. No one can foretell the extent of this disaster.

It is our duty to begin where the Red Cross and other agencies leave off . . . our help should go beyond that, insofar as funds will permit, to assist worthy men to begin anew.

The undersigned invite you to contribute as much as you can. They hope you will respond as quickly and as generously as possible.

Please make use of the enclosed envelope when remitting. Checks should be made payable to the A. V. M. A. Relief Fund.

Thank you.

Fraternally yours,

(Signed) D. M. CAMPBELL
(Signed) J. V. LACROIX
(Signed) H. PRESTON HOSKINS

Any veterinarian who is in immediate need of help, to enable him to resume his professional activities, should communicate with the A. V. M. A. Relief Fund, using the enclosed envelope. Please state your needs in first letter.

A wire to President Foster, attending the Michigan State College Short Course for Veterinarians, at East Lansing, enabled him to lay the situation before the officers of the Michigan State Veterinary Medical Association, which body promptly voted $200.00 from its treasury. This was on January 28. A meeting of the staff of the North Shore Animal Hospital, Evanston, Ill., resulted in a check for $100.00 payable to the A. V. M. A. Relief Fund. This was on the 29th. The same day offers of help, financial and otherwise, were received by local and long distance telephone. The first mail responses arrived on the 30th. Here are a few of the messages which accompanied checks:

More than glad to do this. If this is not enough, will you please call again.—Dr. Reuben Hilty, of Toledo, Ohio, former president of the A. V. M. A.

I am glad to give for those brother veterinarians who are unfortunate in this disaster. No doubt that the extent of damage will not be known until rehabilitation has taken place and if another drive is necessary, another check of like amount can be relied upon from me.—Dr. F. C. Tucker, of Claypool, Ind., immediate past president of the Indiana Veterinary Medical Association.

A grand idea and one that deserves our support.—Dr. D. A. Eastman, of Moline, Ill., secretary of the American Animal Hospital Association.

As outlined in the letter, the plan is to provide financial assistance for veterinarians in the Ohio and Mississippi valleys,

who have been the victims of the recent terrible flood. Although no specific cases of need have been brought to our attention, up to the time that this is being written, it is believed that many veterinarians, along with others in the water-devastated cities and towns along the Ohio River, probably have been heavy losers of property. Even in this territory, where damage by flood is always something of a hazard, very little insurance is carried against loss by flood. Veterinarians probably have had extensive losses of property in the form of drugs, biologics, instruments, books, supplies and equipment. These will have to be replaced just as rapidly as possible, so that veterinarians may be able to render the services so badly needed in their communities. So, briefly, the object of the A. V. M. A. Relief Fund is to provide immediate financial assistance to veterinarians, whether members of the A. V. M. A. or not, who are in need of such help for "getting back on their feet."

The Red Cross and other recognized relief agencies can go only so far in their wonderful work of saving lives and relieving suffering. We, as veterinarians, will step in where these agencies leave off and do what we can to help unfortunate colleagues get back to normal. By so doing we are helping not only our fellow veterinarians but the animal population in the stricken areas as well.

If you did not receive a copy of the letter herein quoted, consider this as your letter. If you are outside of the flood area, send in your contribution. If you are inside of the trouble zone and need help, communicate with the A. V. M. A. at once. If you know of any veterinarian who is in need of help, and who may be too modest to ask for it, let us know about it.

APPLICATIONS FOR MEMBERSHIP

The month of January maintained the pace set by previous months of President Foster's administration in the matter of applications for membership filed. As a result of the activities in this field during January, 43 applications are given first listing this month. This figure is only two below the record for January, established in 1929. For the five months of the present administration the total has reached 202, or an average of slightly over 40 per month.

The impressive fact in connection with the 202 applications received thus far is their wide distribution. An analysis shows that these applications have been filed by veterinarians located

in 38 states, the District of Columbia, Puerto Rico, Canal Zone and Canada. At the same time, the breakdown fails to disclose any one state accounting for a preponderant share of the applicants, as has so often been the case. Last year Ohio had the honors all to herself, and the year before it was Oklahoma that led the procession by a wide margin. In each case the holding of the annual convention in the state was the impelling stimulus.

Ten states have failed to turn in a new member so far this year: Arizona, Delaware, New Hampshire, Nevada, North Dakota, Oregon, Rhode Island, South Carolina, Tennessee and West Virginia. During February a special effort will be put forth to get new members in each of these ten states. This will not be as easy as it looks, as several of these states already have a percentage of A. V. M. A. members considerably above the average of the country as a whole.

(See January, 1937, JOURNAL)

FIRST LISTING

BENTON, CARL R. 139 Vernal St., Everett, Mass.
D. V. M., Michigan State College, 1936
Vouchers: Alden H. Russell and H. W. Jakeman.

BRECHER, CHESTER A. Storm Lake, Iowa
D. V. M., Iowa State College, 1933
Vouchers: Thos. W. Munce and F. B. Young.

BRITTON, WILLIAM S. Britton, Mich.
D. V. M., Michigan State College, 1936
Vouchers: Col. Robert J. Foster and E. C. W. Schubel.

BUNTON, SAMUEL E. JR. Del Rio, Texas
D. V. M., Texas A. & M. College, 1933
Vouchers: Matthew E. Gleason and J. J. Reid.

CHRISTENSON, LOUIE C. Slayton, Minn.
V. M. D., University of Pennsylvania, 1911
Vouchers: William C. Prouse and L. E. Stanton.

CLARK, LT. VIRGIL H. 3103 Hascell St., Omaha, Neb.
D. V. M., Kansas State College, 1932
Vouchers: Capt. M. L. Steele and Lt. Col. Jesse D. Derrick.

COOMBS, ALFRED E. 1 Kennebec St., Skowhegan, Me.
V. M. D., University of Pennsylvania, 1931
Vouchers: J. F. Witter and E. Everett Russell.

COOP, LT. MORAY C. 315 E. Magnolia, San Antonio, Texas
D. V. M., Texas A. & M. College, 1936
Vouchers: Lt. Col. Geo. H. Koon and J. J. Reid.

DEANE, LT. DON L. c/o Veterinary Station Hospital, Fort Bliss, Texas
D. V. M., Colorado State College, 1934
Vouchers: Karl H. Willers and Jean C. Flint.

DEVINE, JOHN J. 2613 N. 29th St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1936
Vouchers: F. E. Lentz and C. J. Marshall.

DICKINSON, RAY T. 2121 Cedar Springs, Dallas, Texas
D. V. M., Texas A. & M. College, 1931
Vouchers: Frank D. Porter and R. P. Marsteller.

EASTERLING, CALVIN H. 407 S. Robert St., Saint Paul, Minn.
D. V. M., Colorado State College, 1920
Vouchers: A. T. Ostendorf and C. P. Fitch.

FRAHM, EDWARD W. c/o Yowell Hotel, McAlester, Okla.
D. V. M., Kansas State College, 1935
Vouchers: C. C. Hisel and L. J. Allen.

FRIES, JOHN A. Durand, Mich.
D. V. M., Grand Rapids Veterinary College, 1913
Vouchers: E. C. W. Schubel and Wm. Hansen.

FUNCHESS, GEORGE T. Box 624, Graham, Texas
D. V. M., Southwestern Veterinary College, 1916
Vouchers: T. O. Booth and A. C. Burns.

FUNCHESS, LUCIUS C. 128 N. Mason St., Bowie, Texas
D. V. M., Southwestern Veterinary College, 1916
Vouchers: T. O. Booth and M. E. Gleason.

GEICK, HAROLD L. 2980 S. Logan Ave., Milwaukee, Wis.
D. V. M., Iowa State College, 1934
Vouchers: F. Kickbusch and B. C. Johnson.

GOLDBERG, LOUIS 2524 Atlantic Ave., Atlantic City, N. J.
D. V. M., Ohio State University, 1923
Vouchers: Chas. J. McAnulty and J. G. Hardenburgh.

GOLDEN, VERNON V. Box 357, Tupelo, Miss.
D. V. M., Colorado State College, 1935
Vouchers: Hartwell Robbins and T. W. Boman.

HERD, DWIGHT G. 132 N. Vine St., Marion, Ohio
D. V. M., Ohio State University, 1935
Vouchers: W. F. Guard and Leonard W. Goss.

IRWIN, HELEN RICHT 538 S. Madison St., Tulsa, Okla.
D. V. M., Kansas State College, 1932
Vouchers: H. Wood Ayers and C. C. Hisel.

IRWIN, WILLIAM F. 538 S. Madison St., Tulsa, Okla.
D. V. M., Kansas State College, 1933
Vouchers: H. Wood Ayers and C. C. Hisel.

KATZ, JULIUS S. Ohio Wildlife Rehearch Station, Ohio State University, Columbus, Ohio
D. V. M., Ohio State University, 1936
Vouchers: W. F. Guard and J. H. Knapp.

KINGMAN, HARRY E. JR. Box 1086, Sacramento, Calif.
D. V. M., Colorado State College, 1933
Vouchers: H. D. Port and H. E. Kingman, Sr.

LYDDY, JAMES D. 941 Hamilton St., Allentown, Pa.
D. V. M., Iowa State College, 1931
Vouchers: R. R. Newman and J. H. Lowe.

MURPHY, JAMES M. 4311 Manayunk Ave., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1935
Vouchers: R. Learmonth and John D. Beck.

NISLEY, LEROY F. Gothenburg, Neb.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: J. W. Murdock and W. T. Spencer.

OPLINGER, ROBERT F. Northampton, Pa.
V. M. D., University of Pennsylvania, 1936
Vouchers: F. E. Lentz and Ira L. Fridirici.

PRCHAL, LT. CHARLES F. 4112 S. 12th St., Omaha, Neb.
D. V. M., Kansas State College, 1935
Vouchers: Lt. Col. Jesse D. Derrick and Capt. M. L. Steele.

PRICE, CAPT. GEORGE T. Fort Sam Houston, Texas
B. V. Sc., Ontario Veterinary College, 1930
Vouchers: Lt. Col. I. O. Gladish and Lt. Col. Geo. H. Koon.

ROBY, LT. ALBERT A. JR. 3815 McKinley, El Paso, Texas
D. V. M., Kansas State College, 1934
Vouchers: Maj. Louis L. Shook and Maj. O. C. Schwalm.

ROSENBERG, ALEXANDER J. 108 Isabella St., Salisbury, Md.
V. M. D., University of Pennsylvania, 1935
Vouchers: Irwin M. Moulthrop and E. J. McLaughlin.

SCHMIDT, CLARENCE P. c/o Veterinary Hospital, Worthington, Minn.
D. V. M., Kansas State College, 1935
Vouchers: Samuel Kelsall, III and A. L. Birch.

SCHWABLAND, JOHN C. Blissfield, Mich.
D. V. M., Michigan State College, 1935
Vouchers: E. C. W. Schubel and Ward Giltner.

SCHWIESOW, LT. CARLTON W. 613 Lynn St., Cadillac, Mich.
D. V. M., Ohio State University, 1936
Vouchers: Capt. B. F. Hudson and Maj. Will C. Griffin.

SERVATIUS, JOHN N. Box 743, Vernon, Texas
M. D. C., Chicago Veterinary College, 1906
Vouchers: T. O. Booth and M. E. Gleason.

SKALA, MATT. J. 3245 E. Sheridan St., Ely, Minn.
D. V. M., Ohio State University, 1934
Vouchers: George E. Jacobi and C. T. Adamson.

SUDHEIMER, ROBERT L. 503 U. S. Courthouse, Fort Worth, Texas
D. V. M., Texas A. & M. College, 1936
Vouchers: A. A. Lenert and H. Schmidt.

TOMSCHE, EMIL J. Albany, Minn.
D. V. M., Iowa State College, 1929
Vouchers: L. E. Stanton and H. D. Bergamn.

TUTTLE, LT. MARTIN L. 2788 Indianola Ave., Columbus, Ohio
D. V. M., Ohio State University, 1933
Vouchers: Maj. A. C. Wight and Maj. F. L. Holycross.

WALTERS, MAURICE K. 310 W. T. Waggoner Bldg., Fort Worth, Texas
D. V. M., Michigan State College, 1934
Vouchers: T. O. Booth and F. C. Steinman.

WERRIN, LT. MILTON Box 1386, Roanoke, Va.
V. M. D., University of Pennsylvania, 1934
Vouchers: Maj. F. H. Woodruff and C. J. Marshall.

WHITNEY, LT. COL. CLIFFORD C. Corozal, Canal Zone
D. V. M., George Washington University, 1914
V. M. D., University of Pennsylvania, 1915
Vouchers: Lt. Col. R. A. Kelser and Col. R. J. Foster.

Applications Pending

SECOND LISTING

(See January, 1937, JOURNAL)

Ackerman, Grant A., Hebron, Neb.
Aichelman, William W., 829 N. Main St., Bellefontaine, Ohio.
Anderson, John S., State House, Lincoln, Neb.
Barbee, James S., Sutton, Neb.
Bolenbaker, Capt. Roger F., 326 Brix Bldg., Fresno, Calif.

Carlson, Fred N., Faulkton, S. Dak.
Cline, Merton L., Plainview, Neb.
Collins, Warnie R., Bancroft, Neb.
Foote, Lt. Bernard E., Hdqrs. Silver City Dist., C. C. C., Silver City, N. M.
Getz, Capt. Austin T., Port of Embarkation, 58th St. & 1st Ave., Brooklyn, N. Y.
Gibson, S. S., Randolph, Neb.
Glenney, William C., Tri-City Animal Hospital, Elgin, Ill.
Hasselbalch, A. E., Saint Edward, Neb.
Herring, Lt. Fred L., United States Military Academy, West Point, N. Y.
Hershberger, Maj. Frank C., Station Veterinarian, Fort Hamilton, N. Y.
Hill, Capt. Verne C., 214 Meade Ave., Fort Leavenworth, Kan.
Lassen, Lt. Keith O., Dist. H, C. C. C., Fort Benning, Ga.
Leonhard, Ernest H., 334 Post Office Bldg., Oklahoma City, Okla.
Lindgren, T. J., Edgar, Neb.
Lovell, Maj. Raymond I., Presidio of Monterey, Calif.
Loy, Carl T., 1303 Drexel St., Omaha, Neb.
Lundberg, Frederick O., Wausa, Neb.
McEachran, I. W., Geneva, Neb.
Mace, Lt. Don L., C. C. C. Hdqrs., Post Office Bldg., Redding, Calif.
Mau, Fred C., 6003 S. Mozart St., Chicago, Ill.
May, Arthur W., 4 Agassiz Park, Jamaica Plain, Mass.
Miller, Roy F., 309 Federal Bldg., Pierre, S. Dak.
Perry, Howard D., Yankton, S. Dak.
Peterson, Lt. Harold O., 8217 19th Ave. N. E., Seattle, Wash.
Phillips, Lt. Harry L., 5th C. C. C. Dist. Base, U. S. Army Wharf, New London, Conn.
Rosenwald, Arnold S., 276 E. Seventh St., Winona, Minn.
Ryland, Joseph H., Box 514, Selma, Ala.
Sandin, Oscar, Plattsmouth, Neb.
Schmela, John H., Norfolk, Neb.
Smith, John W., R. 1, Stockyards Station, Denver, Colo.
Spangler, Don H., 516 University, S. E., Minneapolis, Minn.
Steigleder, Ben H., 4525 Ellis Ave., Chicago, Ill.
Taylor, Irwin A., Durand, Wis.
Thiele, M. I., Prescott, Iowa.
Tudor, Alven O., 334 Post Office Bldg., Oklahoma City, Okla.
Vollmer, Floyd A., Liberty, Ill.
Yount, Virgil A., 334 Post Office Bldg., Oklahoma City, Okla.

The amount which should accompany an application filed this month is $9.58, which covers membership fee and dues to January 1, 1938, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Kansas City Veterinary Association. Baltimore Hotel, Kansas City, Mo. February 16, 1937. Dr. C. C. Foulk, Secretary, 1103 E. 47th St., Kansas City, Mo.

Virginia Polytechnic Institute. Conference for Graduate Veterinarians at. Virginia Polytechnic Institute, Blacksburg, Va. February 16-18, 1937. Dr. I. D. Wilson, Department of Biology, Virginia Polytechnic Institute, Blacksburg, Va.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. February 17, 1937. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

Illinois State Veterinary Medical Association. Hotel Abraham Lincoln, Springfield, Ill. February 18-19, 1937. Dr. C. C. Hastings, Secretary, Williamsville, Ill.

Keystone Veterinary Medical Association. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. February 24, 1937. Dr. M. W. Allam, Secretary, Media, Pa.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. February 24, 1937. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. March 2, 1937. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. March 3, 1937. Dr. R. S. MacKellar, Jr., Secretary, 329 W. 12th St., New York, N. Y.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. March 3, 1937. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

Houston Veterinary Association. Houston, Texas. March 4, 1937. Dr. D. B. Strickler, Secretary, 317 Federal Bldg., Houston, Texas.

East Tennessee Veterinary Medical Society. White Surgical Supply Building, Knoxville, Tenn. March 6, 1937. Dr. Robert L. Hummer, Secretary, 312 W. Church Ave., Knoxville, Tenn.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. March 8, 1937. Dr. J. N. McIlnay, Secretary, 3251 Leavenworth St., Omaha, Neb.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. March 9, 1937. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. March 10, 1937. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Willamette Valley Veterinary Medical Association. Dallas, Ore. March 10, 1937. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

MASTITIS FROM THE STANDPOINT OF A PRACTITIONER*

By C. H. CASE, *Akron, Ohio*

A few years ago, while attending a veterinary conference at Cornell University, Doctors Udall and Fincher inquired as to which phase of cattle practice most urgently required further research. My reply was mastitis. The bromthymol test was soon developed and much has been accomplished since that time, but there is still much that should be done.

At the present time, mastitis causes more loss to dairymen than all of the other diseases of dairy cattle combined. There are many factors which contribute to this loss, one of the chief ones being the decreased value of the affected cows. This is the direct result of their producing milk deficient in both quantity and quality.

The reduced production of one or more infected quarters, when computed for a year, is often surprising. For example, a cow belonging to one of my clients reacted, soon after freshening, to the bromthymol test in both rear quarters; palpation also revealed the presence of nodules in these two quarters. Then, to demonstrate to the owner that infected quarters produce less milk, the milk from the front and rear quarters was drawn and weighed separately. At the end of a week, it was found that the average production of the front quarters was 30 pounds per day, while that of the rear quarters was approximately 25 pounds per day.

Usually the rear quarters produce more milk, so the difference in production between the front and rear halves is actually greater than this 5-pound difference would indicate. But figuring a loss of 5 pounds per day, with milk at 10 cents per quart, we find that the dairyman lost $7.50 per month due solely to mastitis in the two rear quarters of this one cow. In the whole herd, which was recently placed under my supervision, a total of 53 cows are milked. In July, 1936, 29 quarters reacted in various degrees to mastitis tests; 14 of these quarters gave positive reactions to the bromthymol test. It is reasonable to assume that each of these quarters would produce 2 pounds of milk per day less than a healthy quarter; at this rate, the total daily loss in production would amount to 3½ gallons.

When one herd was acquired two years ago, 48 cows were being milked; of these, 24 cows had mastitis—one in all four

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

quarters. Up to this time, their sales of raw milk had steadily declined; some of the consumers even returned some of the milk, due to a noticeable inferiority in quality. The owner was frequently notified by health authorities that his bacteria counts were high. After testing for mastitis and eliminating the infected quarters as they arose, his sales more than doubled and he frequently had counts as low as 100 bacteria per cc. When the herd was sold this year, only three cows showed mastitis.

The significance of high bacteria counts may be more forcibly demonstrated by an illustration. Two years ago, we were called to treat a cow for bloat. While there, the owner said that he had purchased the cow to produce the milk for his family during the summer while they lived on the farm. He and the children, who were consuming all the raw milk, had developed a severe diarrhea; but his wife, who drank no milk, had no such trouble. At first he blamed the water supply, but an examination disclosed nothing harmful there. So each quarter of the cow's udder was tested for mastitis, and two quarters were found to be abnormal; a laboratory examination of samples of milk from the two quarters showed a pronounced streptococcic infection in these same two quarters. Then, after disposing of this cow and purchasing another one that was free from mastitis, their trouble all disappeared within a few days.

Quite often we find that many of the laity have little or no respect for infected milk. Recently, one dairyman complained to us that the city health authorities had prohibited the sale of his milk until his bacteria counts were reduced considerably. Upon examining his herd, one cow was found that gave stringy milk from three quarters. When asked why he sold such milk as that, he replied that any milk which passed through a cheesecloth strainer should be good.

In a similar case, heavily contaminated milk was revealed by an examination of a sample. When the owner was told that his milk contained a million bacteria per cc, he informed us that we could not put that many in a ten-gallon can.

Another time, we were requested to examine two cows for mastitis by a woman who lived near one of my clients. She had purchased these cows a few weeks before at the advice of her dentist, to furnish her child with the milk she required to overcome a lack of calcium in her teeth. My client, knowing that cows purchased so cheaply might be defective, recommended that we make the test. One of the cows was negative to the bromthymol test, and the other gave a positive reaction in both left quarters; a microscopic examination of the milk in the laboratory

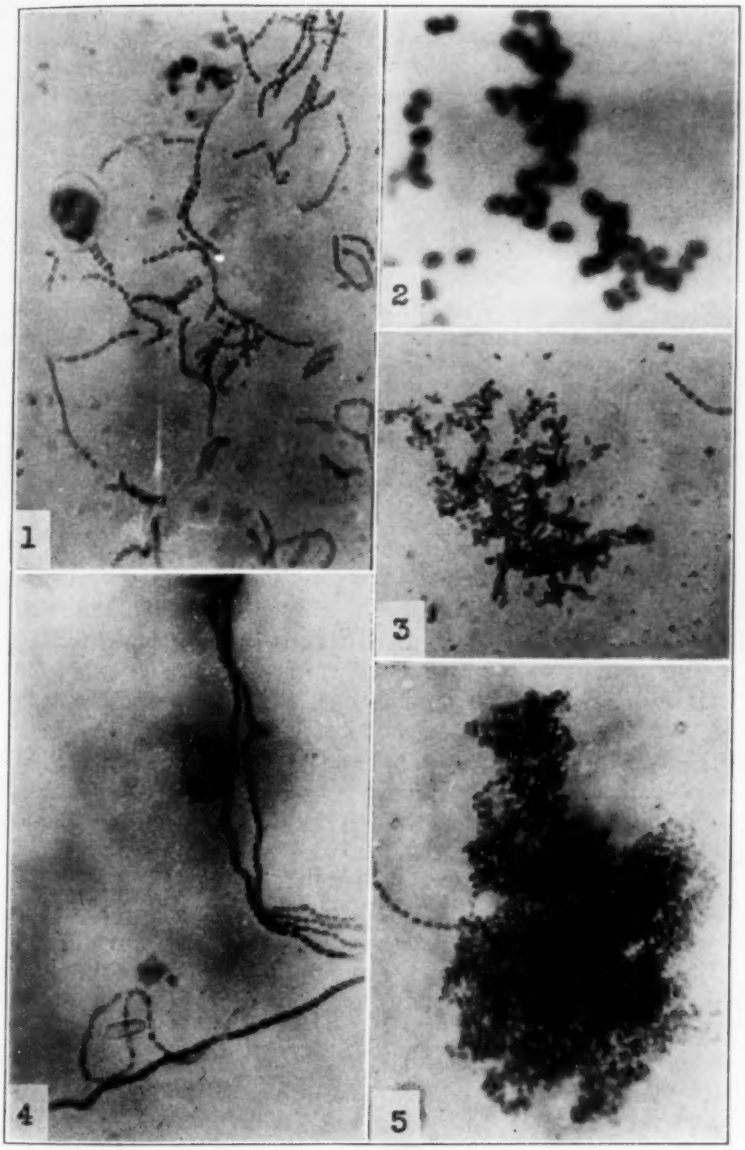

FIG. 1. Short-chain streptococci and leukocytes.
FIG. 2. Diplococci.
FIG. 3. Staphylococci and short-chain streptococci.
FIG. 4. Long-chain streptococci.
FIG. 5. Staphylococci and streptococci.

revealed a severe infection with long-chain streptococci in these quarters. When the cook was advised to discard the milk from these quarters, she objected vigorously, because she had been making such good cottage cheese from the milk from that cow. This milk always soured in twelve hours for her, but the milk from the cow that we found healthy she thought was faulty, because it soured so slowly.

Bacterial Infections Cause Mastitis

Mastitis is caused by a large number of factors—most of them being indirect or predisposing causes. Of the direct causes, bacterial infection of the udder is the outstanding factor. Of all the bacteria, streptococci are the worst offenders; for prognostic reasons, these are still fairly satisfactorily divided into the long- and short-chain groups. In our practice, infections with the long-chain streptococci occur in 13 per cent of the cases of mastitis, as diagnosed by the bromthymol test. Of these infections, most terminate unfavorably.

Similarly, infections with short-chain streptococci occur in 51 per cent of the cases, and usually terminate more favorably with proper treatment. Staphylococci cause a low-grade infection in 39 per cent of our cases of mastitis, and respond more readily to treatment. Diplococci and *Bacillus subtilis* are frequently encountered in bacterial examinations, and are usually considered non-pathogenic contaminants from dust and dirt entering the milk while milking, or from unclean utensils. There is still some question as to the pathogenicity of some diplococci, though. It is our opinion that these infections ordinarily enter the udder through the teat-canal.

The incidence of leukocytes, streptococci and staphylococci in diseased samples of milk, as detected by the bromthymol and microscopic tests in our herds during 1934-35, is shown in table I.

From this table, it can be seen that leukocytes occur in all the samples of milk, although the number may be very low. The discrepancies in the numbers given for the range of "few" or "many" is unimportant, and will vary with the examiner. Here it is more important to be consistent with any one numerical range than it is to emphasize the limits of this range, as the number is relative anyway, and serves only to indicate the change from one sample to the next. These ranges were selected by directly examining prepared mounts of milk through the oil-immersion lens of a microscope calibrated so that one object in a field would be equivalent to about 6,000 of these objects per

cubic centimeter of the milk. The technic of preparing these mounts will be explained later.

Injuries are frequently another direct cause of mastitis. These result in an influx of leukocytes into the affected area, which is usually easily detected by a microscopic examination of the milk. Bacterial infection may follow later. Injuries may also be an indirect cause of mastitis, as when a cow steps upon a teat in getting up. In healing, connective tissue induration may cause a constriction in the teat-canal—commonly known as a "spider." Then, if the milker is not especially careful to milk that quarter dry each time, infection is liable to result.

TABLE I—*Incidence of leukocytes and bacteria in samples of milk.*

	PER FIELD	SAMPLES	
		No.	%
Leukocytes	Few (10 or less)	677	52
	Many (11 to 30)*	445	34
	Lots (more than 30)*	185	14
Short-chain streptococci	Few (5 or less)	300	23
	Many (6 to 30)	202	16
	Lots (more than 30)	157	12
Long-chain streptococci	Few (3 or less)	80	6
	Many (4 to 30)	63	5
	Lots (more than 30)	23	2
Staphylococci	Few (3 or less)	350	39
	Many (4 to 30)	141	26
	Lots (more than 30)	27	11

*Many or lots of leukocytes in a sample, accompanied by no bacteria, occurred in 161 (12%) of the cases.

INDIRECT CAUSES OF MASTITIS

Dirty milking-tubes: Among the many indirect causes of mastitis is the promiscuous use of milking-tubes and teat dilators. These are often used in an attempt to cause a teat that contains a "spider" to be milked more easily, and frequently, sufficient asepsis is not exercised. In these cases, it is better to lance the stricture sufficiently to allow the milk to be drawn with ease, and then instruct the owner to milk that quarter as usual after it heals.

Unclean stalls: Lack of bedding and insanitary stables are other factors causing mastitis. When a cow with an udder full of milk is lying down, the pressure on the lower quarters may

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force their sphincters partially open—thereby allowing infection to enter the teat-canal more easily. If the stall is wet and dirty, infections are much more liable to follow.

Careless milkers: Many milkers would undoubtedly be bigger successes in other lines of work. Some who take great pride in demonstrating their speed are frequently careless. One of my clients employed a man who proclaimed himself the fastest milker in the county, so the owner started this man to milking eight cows twice daily. He proved his speed by milking his eight cows while the others milked only five or six. For two years, none of these cows had mastitis, but at my next monthly test we found that five of his eight cows had developed mastitis in one or more quarters. He received his check that night. A month later, the other milkers had most of the quarters back to normal again. Such cases demonstrate the necessity of milking each quarter dry at every milking. More care should be used when a stranger is milking the cow, especially if the cow has a nervous temperament. Such cows may withhold much of their milk, causing much variation in the amounts given at each milking. If this continues very long, mastitis may follow.

Improper drying-off: The improper drying-off of cows is a common causative factor in mastitis before and after the drying period; if any are positive for mastitis, they should be milked dry two or three times daily to freshening time, or else a calf should be allowed to nurse these quarters dry until the cow freshens. Many quarters could be saved if this was done. In one instance that was brought to my attention, a professor from the Extension Department of our Agricultural College lectured to a group of dairymen in one community. Upon being asked by two of my clients as to which was the best method of drying off dairy cattle, he used the university herd as an example where this was accomplished merely by an abrupt cessation of milking; his inference was that the milk would be absorbed. So, two cows, which were previously free from mastitis, were selected for the trial. When the first cow calved, two quarters were infected with streptococci, and were subsequently lost. The other cow similarly lost one quarter after freshening. The loss in value of each cow was sufficient to impress these dairymen adequately that cows should not be dried off in this manner. Some dairymen object to milking continuously up to the next lactation period because the amount of milk produced for that period will be reduced, but if the health of one or more quarters is dependent upon this procedure, its worth is amply justified.

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Milking-machines: Many cows develop mastitis from the use of milking-machines. These may be used safely only where great care and sanitation are exercised. Very few herds can produce certified milk if milking-machines are used, due to the inevitable rise in the bacteria count beyond the limit of 10,000 per cubic centimeter. Mastitis is spread more easily through their use also. Of all the herds which we inspect each month, the one having the most trouble with mastitis is, and has been, using a milking-machine.

Excessive nursing: Many cases of mastitis in heifers that have just freshened are caused by their being sucked while they are quartered with other calves. If this vice continues, enough milk may be secreted to cause a marked development of the udder. Then, when the calves are separated, the excessive amount of milk retained in the udder may lead to infection in one or more quarters. At freshening time, these infected quarters may have become totally destroyed, producing no milk whatever, or partially incapacitated, so that only a small amount of milk will be secreted. One example will illustrate what can be done with these cases if handled properly.

About a year ago, we were called to treat a heifer 18 months old, for a badly caked udder. An examination showed that it contained much milk, although the heifer had never been bred. The owner was instructed to milk her dry, twice daily. The next day, she was turned out into the lot as usual, which she shared with about 80 lambs. In a short time, the owner noticed that two of the lambs approached the heifer and nursed with great gusto, enjoying perfect coöperation from the heifer. The mystery was satisfactorily solved. The owner continued to milk her twice daily until she freshened. Now, in the early part of her first, normal lactation period, she is producing four gallons of milk daily from a healthy udder. If she had received no attention before freshening, the udder would undoubtedly have been destroyed.

Early Detection of Mastitis

The importance of detecting mastitis in the early stages of the disease cannot be overemphasized. In many cases, a favorable termination of the disease can be expected only when this is done, and the proper treatment is instituted.

Of the various methods we have tried for the detection of mastitis in the field, none have proved quite so satisfactory as the bromthymol test. For simplicity, accuracy, rapidity, and the impression it conveys to the dairymen, who can see a color reac-

tion, it is hard to surpass. When this test is followed by a microscopic examination of the milk in the laboratory, an adequate picture of the pathological processes within the udder is usually presented.

Bromthymol Test

The practical application of the bromthymol test is not difficult. The basis of this test is the bromthymol solution, which indicates, by color changes, the degree of alkalinity of the sample of milk tested. This solution was formerly quite expensive when it was made with alcohol, but an equally efficient and relatively inexpensive, aqueous solution was developed by Dr. C. E. Hayden, of Cornell University, for which we are very grateful. Using the following formula, the cost of this solution is about $4.50 per gallon, or about 1½ cents per sample tested:

℞

Bromthymol blue powder	5.0 gm
Sodium hydroxide solution (5%)	10.0 cc
Dilute hydrochloric acid	q. s.
Distilled water, q. s.	3,750.0 cc

Directions: Make a solution by first adding the 10 cc of sodium hydroxide solution to 800 cc of distilled water. Completely dissolve the 5 grams of bromthymol blue powder in this solution by shaking in a gallon bottle. Then add the distilled water to make 3,750 cc. Finally, add the dilute hydrochloric acid, a drop at a time, stirring or shaking between drops; continue until a uniform sherry color is reached. If the solution becomes too light in color, a small amount of the sodium hydroxide solution will darken it again.

Of this solution, we use 1.5 cc for each 5 cc of milk to be tested. Test-tubes of about 20 cc capacity are marked with a file at the level of 5 cc of solution contained therein. Three or four sets of four such tubes are ordinarily used at most dairies, that is, one set for each person collecting the samples. Various types of kits have been devised for carrying these tubes, along with a bottle of the bromthymol solution, a pipette for measuring the solution, a test-tube brush, and a few sterile test-tubes in which milk samples are collected for examination at the laboratory.

To conduct the test, give each milker a set of four tubes, which he holds in his left hand. To insure a more representative sample from each quarter, conduct this test just before milking time, and discard the first two or three streams of milk from each teat into the gutter or a suitable receptacle. Then, for the sake of uniformity, the milker draws 5 cc from the left front quarter first into the tube at the end of his thumb; then 5 cc from the left rear quarter into the next tube, 5 cc from the right rear quarter into the third tube, and finally 5 cc from the right front quarter is milked into the last tube, which is held at the junction

of the thumb and forefinger. He brings them to you in this order, and you add 1.5 cc of the bromthymol solution to each tube of milk. Then he places his finger over the end of each tube, and shakes each a time or two so that a uniform mixture will be made. In doing this, it is necessary to retain the same order of the tubes until you note the reaction.

The presence of mastitis in any quarter is then detected by noting any deviation in color of its respective tube of milk from the yellowish drab of normal milk. Supposedly, the more pronounced the mastitis is, the more alkaline the milk will become, which is normally slightly acid in reaction; and actually, as the alkalinity of the milk increases, the greener will the bromthymol solution turn it. This alkalinity of the milk is caused by the escape of blood-serum, which is alkaline, into the milk during the process of the disease. A leukocytosis of the milk, which follows most injuries of the udder, will also show a similar reaction. The degree of reaction of each test is then recorded, using the following code:

— normal; yellowish green.
T trace of a reaction; darker than normal.
P partial reaction; bluish green.
+ positive reaction; dark green.

Some cows that are in an advanced stage of lactation will consistently show a trace of a reaction. Variable reactions occur with the kinds and stages of infections or injuries of the udder.

How Records Are Kept

Our records are kept in a loose-leaf notebook, the sheets being ruled for twelve months. These are very satisfactory for this purpose. The book we use is a product of the Wilson-Jones Company and is a slotted, lock binder, style SS, and form 1284. The leaves are a product of the same company, and are form 84713. We purchase these, with an alphabetical index, from any office-supply store.

For each quarter that shows an abnormal reaction, we collect a sample of milk in one of the sterile tubes for examination at the laboratory, if this was not done during a previous test. Until we report the results of this examination to the dairyman a day or two later, he is advised to refrain from using the milk for human consumption.

Laboratory Examination

The technic which we use in examining these samples in the laboratory is as follows: Place the tubes of milk in an incubator

at 37.5° C. for twelve hours. Then, for each sample of milk, place .01 cc upon a sterile, glass, microscope slide; this is measured by a small pipette marked at .01 cc, and which may be purchased from the Kauffman-Lattimer Company, in Columbus, Ohio. An ordinary slide (1 x 3 inches) will easily hold four such mounts. Before each sample is taken, the pipette should be rinsed, first in distilled water and then in alcohol, to prevent any contamination of the following mount. After each mount is made, it should be spread over one square centimeter of the slide with a wire bent at right angles, with the base of the L thus formed being nearly 1 cm long. A wire loop used in bacteriological work may be easily converted into this instrument. Glass slides with areas one centimeter square etched upon them are very convenient for this procedure. When each mount is spread properly, sterilize the wire spreader by holding it in a gas flame until it is red hot; allow it to cool before spreading another sample. The mount is then fixed by heat, either by carefully passing it over a gas flame, or by placing the slide upon a hot-plate or over a hot, electric bulb. When it has dried sufficiently, allow it to cool and then flood it for two or three minutes with Newman's stain, which may be prepared according to the following formula, which is taken from Udall's "Practice of Veterinary Medicine":

℞

Methylene blue powder	1 to 1.5 gm
Ethyl alcohol (95%)	54.0 cc
Technical tetrachlorethane (Eastman Kodak Co.)	40.0 cc
Glacial acetic acid	6.0 cc

Add alcohol to the tetrachlorethane in a flask and heat to a temperature not to exceed 70° C. Add combined solution to powdered methylene blue. Shake vigorously until the dye is completely dissolved. Cool the solution and then add the glacial acetic acid very slowly. Then wash the slide in water until the mounts become a light blue, and dry. They are now ready to be examined under the oil-immersion lens of a microscope.

The results of this examination are recorded upon the back side of the sheet upon which the corresponding results of the bromthymol test were noted; thus, a complete record is always available instantly. In recording the results of this microscopic examination, the identification number of the cow is noted, the quarter from which the sample was taken, the date, the degree of leukocytosis and the relative number of each kind of bacteria as shown in table I. A copy of this report is mailed to the dairyman, with instructions concerning the proper disposal of the milk from that quarter and the therapeutic measures to be taken.

Interpretation of Results

In general, the following interpretations of the results of such examinations have proved very satisfactory. About 12 per cent of these samples show nothing more than a great increase in the number of leukocytes; these we attribute to injury and such quarters usually become normal within a few days. Milk from these quarters is safe for human consumption. Any number of *Bacillus subtilis* or diplococci we consider as harmless contaminants. When only a few staphylococci or short-chain streptococci are present, the dairyman is advised to discard the first one or two cupfuls of milk from that quarter and use the rest, as the newly secreted milk contains very few bacteria.

If long-chain streptococci, or many or lots of staphylococci or short-chain streptococci are present, a diagnosis of infectious mastitis is made, and the milk is not used for human consumption until it becomes negative to a subsequent examination. Such milk is safely fed to calves or pigs, so it is not necessary to throw it away. There is no direct communication between the various quarters of an udder, so in most cases of mastitis, there should be no fear that an infection in one quarter will necessarily precede trouble in another quarter; a severe infection may remain in one quarter for months without ever spreading to another quarter in that udder.

Prophylactic Measures

As in all other diseases, prevention is the best policy. All probable, etiological factors should be removed as far as possible, especially if treatment is to be of any avail. Dipping the teats in a chlorine solution and sealing their ends is a measure which promises to prevent infections through that channel. Another good measure is to segregate all the infected cows, if possible, and milk them last; of course, all infected quarters should always be milked after the healthy quarters. The liberal use of antiseptic solutions also helps to control mastitis; for this purpose we use HTH(65) in 0.5 per cent solution. This may be purchased from the Mathieson Alkali Works, Inc., 60 East 42nd Street, New York City. This solution is relatively cheap, and if it is used reasonably fresh, it is very efficient. Mastitis may be almost eradicated from a herd by selling the infected cows when they are no longer profitable, and properly raising heifers to replace them.

Treatment is most successful in those cases which are detected early. For that reason, a monthly examination should be con-

ducted in the field—testing the milk with bromthymol solution and palpating the udders for nodules—followed by a microscopic examination of all milk samples suspicious or positive to the bromthymol test. From this an accurate diagnosis can be made, and the necessary therapeutic measures instituted before the disease becomes chronic.

Treatment

One of the best treatments for infectious mastitis is to strip the infected quarter thoroughly dry at least twice daily; oftener is frequently more beneficial. This procedure is followed regardless of the stage of lactation, if necessary, as has been previously mentioned. By using this treatment alone, many milkers can effect a cure in one or two months, even though many streptococci were found in that quarter upon the microscopic examination.

Certain cows are charged to one milker, who strips the affected quarters out thoroughly with one hand while he kneads and massages the udder with the other hand. In acute cases, this tends to break down the nodules which may have formed within the gland. These nodules are really abscesses in the udder, which occasionally break, discharging much pus into the milk. This usually causes the milk to become very stringy. When the abscesses heal, the milk appears normal again. This same treatment is frequently carried out more economically and just as satisfactorily by allowing calves to strip and massage these quarters. Many good calves have been raised upon such milk, and of those we have had the opportunity to observe as cows, none have developed mastitis.

Some acute cases are benefited by rubbing white liniment into the udder. Here, too, frequently milking is the best treatment. Chronic cases are seldom cured by any treatment. In many cases, there is apparently some cryptic factor present which causes an infection to subside or vanish at a succeeding period of lactation, if the cow was dried off properly. For example, our records show that one purebred Holstein had a streptococcic infection of a year's standing in one quarter. This quarter was atrophied, and produced very little milk. At the close of that lactation period, two calves were placed in the box-stall with her until freshening time, when the infected quarter resumed a normal secretion of milk again, but it still contained a few streptococci. So, four calves were placed in her stall for two weeks after she freshened, and the milk became normal. She was then placed in the healthy herd again, where she produced over 100

pounds of milk daily for a month. She has remained healthy for the last three years.

Sure cures for mastitis warrant just about the same value as do those for any other chronic disease. As may be seen from table I, one-eighth of the cows reacting to the bromthymol test in our practice reveal nothing but a leukocytosis of the milk, which usually indicates an injury to the udder. If such a cow is injected with some proprietary preparation, and ceases to react to the bromthymol test, the agent is given full credit for the cure, regardless of the fact that the udder would have healed within a few days anyway, just as most bruises do. A true case of infectious mastitis is cured only when aseptic milk is being produced continuously from that quarter, and this can be determined only by a bacteriological examination of the milk.

One of the most important factors in the control of mastitis is the prevention of the disease. Another factor, which is of doubtful value, is the Federal Plan as advanced by the government recently. A measure which is very worthy was adopted by the Ohio Guernsey Association several years ago and requires that all cows sold at the state sale must be guaranteed free from mastitis; no cow reacting to the bromthymol test the night before the sale is sold. In this respect, mastitis will eventually be placed upon the same par as Bang's disease and tuberculosis.

Many beliefs prevail within the profession as well as among dairymen, that have little, if any, foundation. For instance, in some cities, even ordinances have been passed which prohibit the sale of milk from a cow that has mastitis in one or more quarters; such a cow is required to be sold or quarantined. According to our records, which date from the fall of 1931, such a ruling is unfair to the dairyman. If mastitis exists in only one quarter, the milk from the remaining three quarters is just as safe as milk from a cow with four healthy quarters. A similar belief infers that if mastitis exists in one quarter, infection of another quarter will soon follow; there is practically no foundation for this inference.

Our records show that of 201 cows out of the 247 cows definitely infected with mastitis in one quarter, none developed an infection in another quarter that reacted to the bromthymol test for three months in succession. Only 46 (19%) reacted to the bromthymol test in another quarter for three months in succession, with most of these (56.5%) recovering in three months or less in the second infected quarter. Similarly, of 119 cows that had one or more quarters totally destroyed, only 7 (5.9%) suf-

fered the loss of another quarter. The rules governing the production of certified require milk from all four quarters; considering the above data, the necessity for such a requirement is doubtful.

The production of good, raw milk should be the goal of every dairyman. With proper coöperation from his veterinarian, he should seldom fail. With the veterinarian periodically testing for mastitis as previously explained, incidental with his other work, the dairyman is then well able to produce almost perfect milk which may be best consumed raw. The average bacteria counts on all the milk produced for consumption in Akron for the year 1935, as found by the Akron Board of Health laboratory, are shown in table II.

TABLE II—*Average bacteria counts of milk produced for consumption in Akron during 1935.*

MILK	BACTERIA PER CC
Pasteurized (standard)	19,106
Grade A pasteurized	11,351
Grade A raw	7,783
Certified	4,362

From table II, one can easily deduce that good raw milk is more wholesome than good pasteurized milk. With adequate veterinary assistance, a dairyman should reasonably expect to produce raw milk well under the limits for bacteria counts imposed by most cities. As an example, one of my clients is milking a herd of 25 cows, upon which we conduct a complete mastitis test each week. The average, weekly, bacteria count of the milk produced from this herd for 1935, as found by the Akron Board of Health laboratory, was 953 per cc. So far, we do not know of another herd in the United States that has done so well. Samples of this milk have remained just as sweet at the end of two weeks in the refrigerator as when it was first produced. A fresh, bottled sample had a bacteria count of 300 per cc, and after being in the refrigerator for this two-week period, the count increased to only 3,000 per cc. The production of milk with such a low bacteria count would not have been possible if a weekly, bromthymol test had not been conducted. This detected cases of incipient mastitis long before they would have been recognized by any other means. Such quarters were eliminated before they affected the bacteria count.

In producing milk with low bacteria counts, a complete record of all reactions to the tests for mastitis must be maintained. A summary of our records reveals that only one herd, of the 35 under our supervision during 1936, has not shown a single, positive reaction to the bromthymol test for mastitis to date. Further examinations of these records show that during the years 1934-35, 2,616 cows were tested for mastitis. Of these, 247 (9.44%) reacted to the bromthymol test in one or more quarters for a period of three successive months. These we consider had mastitis. Stated more simply, approximately one cow, of every ten tested, had mastitis. Similarly, of these 2,616 cows tested, one cow in 22 had one or more quarters which were totally destroyed. During the two years, we find that 1,307 samples of milk were examined in the laboratory for mastitis. This averages almost exactly one sample for every two cows tested during a year. This year, we have been conducting monthly tests for mastitis upon about 1,200 cows. About half of these are producing certified or Grade A milk, the others producing standard milk. The bacteria counts on any of this milk from any herd seldom approaches the 20,000 limit established by the City of Akron.

The City of Cleveland has created an incentive for dairymen producing Guernsey milk in that area to produce better milk; that is, the producers are paid a premium of 10 cents per hundred pounds on all their milk which contains fewer than 15,000 bacteria per cc. Only 5 cents per hundred pounds is paid if the bacteria count averages between 15,000 and 20,000 per cc.

Dairies exist around all cities, and it should be the duty of each veterinarian in these communities to assist them to produce better milk. All that is necessary, in many cases, is to make them aware of the fact that you are in a position to do this work—they already know the value of it. Such work mutually benefits everyone—the veterinarian creating more business for himself; the dairyman producing more milk of a superior quality; and the public gets a better product that may safely be consumed raw, as nature intended.

Discussion

Dr. Silas Kempf: We used to have a good deal of mastitis, which proved to be a feed trouble, possibly a forage poisoning. I should like to ask the doctor what his treatment is when he has any of that kind of trouble.

Dr. Case: You mean acute cases where the udder swells up very large and secretes a yellow, watery kind of milk?

Dr. Kempf: Yes, sir. It does so, sometimes, just over night.

DR. CASE: I do not know the cause of this form of mastitis. I have thought a great many times it was wild cherry. It generally occurs in the summer time when the cattle are out on pasture, in dry weather. They come in in the morning and the udder is as hard as a rock. A great many do not show any bacteria when you first examine the milk. A great many of them have an enormous number of leukocytes. As a rule, afterward they show streptococci. I do not know the cause of it.

What we do is to get those cows in and foment them every hour with hot packs and then cold packs. Change them back and forth and rub them and work them and get the milk out. If there is a good flow of milk, oftentimes the secretion gets started again and they will come back. You lose a great many quarters of that kind. I wish somebody would tell me what they get hold of. The cows are milking right along normally.

DR. M. J. JONES: In most cases I have found that a good laxative or cathartic, followed by nitrate of potash, given about three hours apart, until three doses are given, is beneficial, and with warm applications to the udder, those cases are usually all right in 24 hours. I consider it due to an acid condition in the milk, when they have been browsing on acid plants at different times.

DR. G. M. POTTER: What percentage of cows have you found it necessary to destroy as incurable?

DR. CASE: It depends upon what the cow is. If the cow has three quarters, she may give enough milk out of those three quarters to pay for her feed. It all depends upon the amount of milk she gives. As to a pure-bred cow, with value as a breeding animal for the reproduction of the kind we want, we keep her although she may have only two quarters.

Referring to this chart, this cow was a valuable cow. She came in at the first freshening. We were unable to break the nodules open and keep them open. She lost both of those quarters. She has been going on that way and has been that way since she first freshened. The destruction of those cows depends upon what they produce and what they are worth as pure-breds.

DR. L. A. MERILLAT: Is there any relation as far as the particular quarters that are affected?

DR. CASE: No, none. It does not seem to make any difference.

DR. MERILLAT: Your figures do not show right or front or left or right.

DR. CASE: The most infections would be in the rear quarters, either right or left.

DR. MERILLAT: Have you any theory as to why that is?

DR. CASE: No. Of course, the barn should be built for the length of the cow. Barns are always built the same, and if you have a long cow, when she lies down she lies with her rear quarters in the gutter where she is in more danger of infection. I think that has had something to do with it. With the pressure of lying down, maybe with a large udder lying over into the drop, there is more danger of infection getting in there.

DR. POTTER: In cows that are showing temperature, for how long a period do you withhold them from production?

DR. CASE: Those cows generally show a dark reaction and I tell them to use the milk for calf feed until it clears up. A good many dairymen have their own testing outfit, and they test every day to see that the cow gets back into the milking barn. A cow with high temperature should not be used.

DR. D. M. PHILLIPS: I have quite a number of cases similar to those the brother over here just mentioned. I have been anxious to know whether there is any intravenous treatment for those cases.

DR. CASE: I have tried metaphen (1:2,000) intravenously, and I have given bacterins, and I made autogenous vaccines from the milk in a kind of crude way, and they just keep on. When you examine the milk under the microscope, it still shows bacteria, and it does not give any results at all. I tried different proprietary vaccines and I have not got anywhere at all. I used to think I got results because the milk seemed to look better, but when you use the bromthymol test every month, that tells you whether you get any results or not.

You see, this whole scheme that I have tried to develop to make good, clean milk is to test the cows regularly every month and watch them. You can detect these conditions long before they know about it, and then get a good milker to get all that milk out. A lot of cows are hard milkers in one quarter and if you have a fellow who is careless, he will quit milking and leave milk in that quarter, and the first thing you know you have a quarter off. You find many of those quarters that react to bromthymol, and that is the history back of them.

DR. F. W. GRAVES: Do you find any correlation between Bang's disease and mastitis?

DR. CASE: No.

DR. GRAVES: What consideration do you give the bromthymol test on a cow after five-eight day freshening?

DR. CASE: I make the bromthymol test on a cow that has just freshened, and if her udder is caked and she shows a reaction in one or more quarters I tell the owner to watch that quarter, milking often and I take a sample of the milk to the laboratory to examine it under the microscope and report back to the owner if the quarters show infection with bacteria or leukocytes.

DR. GRAVES: Do you put the cow back in the milk line by the tenth day?

DR. CASE: Whenever the milk shows a normal reaction to the bromthymol test.

DR. GRAVES: Would you consider as unsafe milk with this 20,000,000 leukocyte count without the presence of streptococci or phagocytosis?

DR. CASE: Oh, no. I have had only one herd of all the 35 herds I take care of that has had a perfect record so far this year, except this one cow, Number 6. When I went to the farm to make the test, the herdsman told me, "This cow is a very valuable pure-bred Guernsey, but she has a habit of running like a horse every now and then." This cow has a record of over 900 pounds of fat. When that bag swings, the milk flies in every direction. That morning the herdsman saw her start to run. He knew I was coming, so he tried to get her into the barn. She fell down and we got a reaction to the bromthymol test in one quarter which showed 20,000,000 leukocytes. The next month the quarter was normal.

DR. GRAVES: Dr. Case, I think you are to be congratulated on this. I happen to be in public health work. The work you have done here is almost an exact duplicate of what I have seen in following veterinarians where we have had epidemiological investigations, or something of that type. It is a thing that every veterinarian can do. You are one hundred per cent sound, and, as I see it in New York state, unless the veterinarians of that state become more interested in this type of work that Dr. Case is doing, you are doomed to lose a lot of business that belongs to you. I think I have never listened to a paper or to a discussion that has come closer to hitting the bottom of this mastitis program than what Dr. Case has given. You are to be congratulated. (Applause)

PYELONEPHRITIS OF CATTLE AND HORSES*

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Of the inflammatory nephropathies affecting the female bovine, pyelonephritis is by far the most common. Pyelonephritis has long been known to European veterinarians, but its presence in the United States was not reported until 1918,[1] at which time it was regarded more or less as a curiosity. Today the infection is known to exist in practically all sections of the country where cattle breeding is an important industry.

Jones and Little,[2] of the Rockefeller Institute, were the first American veterinarians to investigate the disease carefully, and in 1925 they published an article entitled, "Specific Infectious Cystitis and Pyelonephritis of Cows." Udall,[3] in discussing the frequency of the disease, writes as follows:

> Pyelonephritis of cows has been infrequent in our clinic until within the past eight years. From 1915 to 1924, not a single case was diagnosed. In the eight-year period from July, 1924, to June, 1932, 34 cases were recognized and twelve of these were in the year 1931-32.

In our clinic, the disease in the adult cow has become so common that it is from an economic standpoint regarded as highly important. We do not recall having encountered the infection in the mare prior to 1929, but since that period we have studied the disease in seven mares and one stallion.

The affection, when occurring in the mare, presents the same general clinical characteristics as are observed in the cow. Bacteriological and chemical studies of the urine also show a very marked similarity. The presence of the disease is usually not recognized until shortly following parturition, and for this reason has given rise to the belief that the infection is associated with diseases affecting the reproductive organs, especially suppurative inflammatory conditions involving the uterus. While the manner in which natural infection occurs has not definitely been determined, we believe it is usually urogenous and seldom hematogenous in origin. This opinion is based upon the fact that we have definitely shown that the causative organism is usually not associated with vaginal or uterine infections and that the disease is no more frequent in the cow that has thus been affected than in the patient that has experienced a normal parturition and

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whose genital tract has remained free of disease. Pyelonephritis may also be encountered in unbred heifers and fillies, but this is the exception rather than the rule.

The urogenous origin of infection is supported by the fact that the bladder and ureters are involved as well as the kidneys proper and, furthermore, structural changes occurring in the glomeruli, such as exist in embolic nephritis of hematogenous origin, are absent.

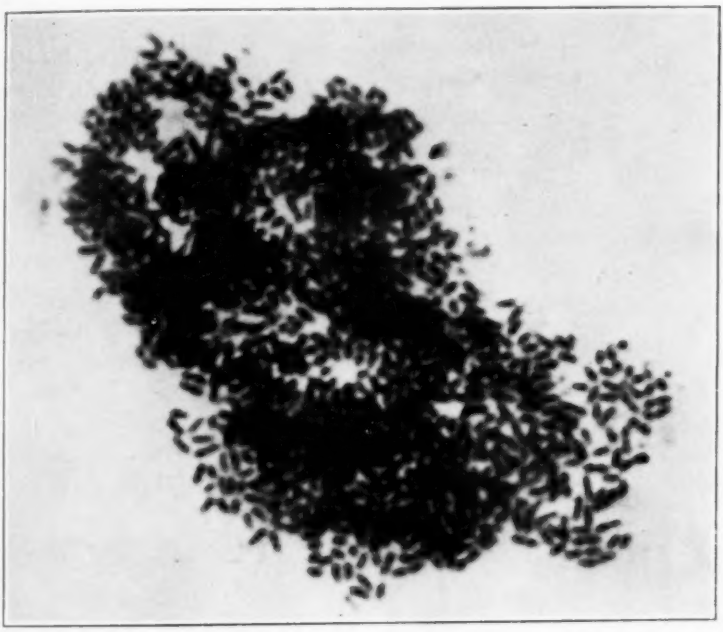

FIG. 1. Characteristic clumps of corynebacteria as found in stained smears of urine sediment.

ETIOLOGY

Corynebacteria have been isolated from the urine of all eight cases of pyelonephritis observed in horses. These organisms have been a characteristic feature of the disease and answer the description of *Corynebacterium renale* (Bergey[4]), as observed in pyelonephritis of cattle. The Corynebacteria were constantly found in characteristic clumps in stained smears of urine (fig. 1). In many cases it was not necessary to concentrate the sediment in order to demonstrate them, as they were present in such large numbers.

Studies of smears of the urine of bovines have demonstrated, from time to time, the presence of streptococci, cocci and strepto-

bacilli, as well as Corynebacteria. These organisms have been noted also by Höflich,[5] Gair,[6] Bemis,[7] Jones and Little[2] and others. The occurrence of these additional organisms was a constant finding in the sediment of urine samples from horses. Figure 2 demonstrates the characteristic picture found. The most striking feature of the picture is the large cocci in long-twisted chains. Sometimes there was a reduction of the chains to an amorphous mass containing cocci and Corynebacteria. There

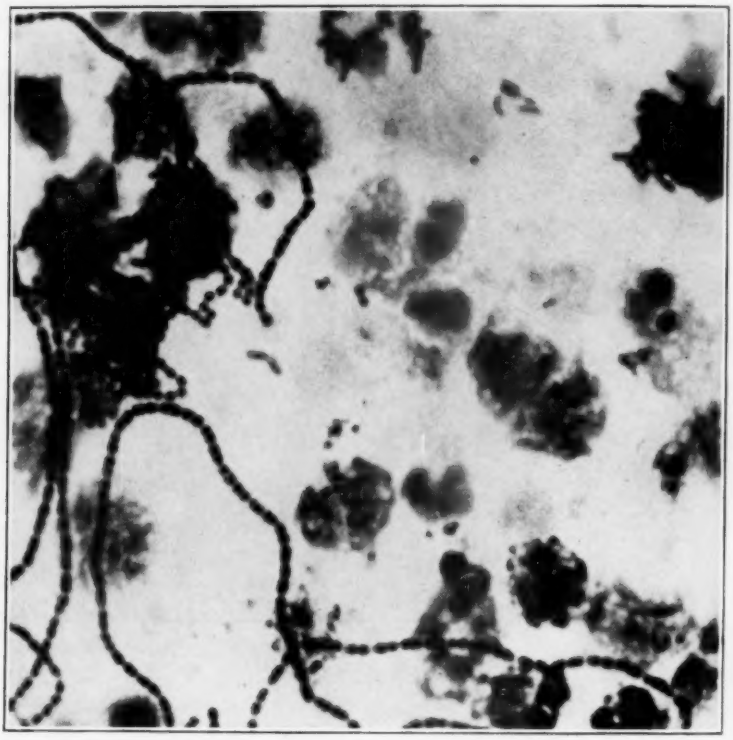

FIG 2. Stained smear of urine sediment. Note long-chained streptococci, corynebacteria and pus cells.

usually were present also short-chained cocci. The cocci in these chains were about one-third the size of the cocci in the long chains. Many intermediate forms of organisms also were found and many pleomorphic forms of what appeared to be Corynebacteria.

It is difficult to say just what relation the streptococcus has to the Corynebacterium, and also what part it plays, if any, in the production of the disease. The Corynebacteria are a constant

feature of the disease in both cattle and horses. Cases have been reported also of finding these same organisms in pyelonephritis of other animals, *i. e.*, dogs, swine and sheep. Jones and Little[8, 9] have been able to reproduce pyelonephritis experimentally in cattle with this organism.

The streptococcus found so regularly in the equine cases and frequently in the bovine cases may easily be a secondary invader although it is a very constant feature. It is thought, however, that this organism may be the result of the growth of the Corynebacteria over many generations in alkaline urine and is a dissociated form of it. Many authors who write on dissociation of streptococci point out the diphtheroid as one of the dissociating forms and also note a close relationship between these two organisms. Many times direct culture from the urine failed to yield streptococci, whereas the Corynebacteria were isolated easily. The Corynebacteria remained constant in their biochemical reactions, the streptococci were not constant in this respect, and no correlation could be made between streptococci isolated from different cases.

SYMPTOMS

The patient exhibits urinary disturbances that are characteristic of the disease. The frequent passage of bloody urine is quite pathognomonic, and usually attracts the attention of the owner or caretaker before other symptoms are noted. The urine at times may vary from clear to cloudy, and frequently contains mucous clots. Small bright red blood clots appearing at completion of evacuation of the bladder indicate the occurrence of recent hemorrhages. Other symptoms consist of progressive emaciation, inappetence, and decreased lactation. As the disease is usually chronic, loss of flesh is very slow. In the advanced cases pronounced symptoms of colic are frequently observed. Especially is this true of the equine. Incontinence of urine in the mare as the result of extensive inflammation of the bladder is quite common. The urine thus voided produces a denuded area of the skin immediately below the vulva.

DIAGNOSIS

Diagnosis is dependent upon a chemical and bacteriological examination of the urine together with a careful physical examination of the bladder and ureters. Palpation of the kidneys also should be attempted. Chemical analysis of the urine shows the presence of albumin. Microscopic examinations of the sediment reveal the presence of erythrocytes, leukocytes, diphtheroid bacilli, and at times streptococci. Blood examination shows that

there is retention of urea. One skilled in conducting rectal examinations in the cow can quite easily determine the extent of the disease, particularly when there is marked thickening and contraction of the bladder and extensive dilation of one or both ureters, particularly the left ureter and kidney. By careful manipulation one may detect the presence of adhesions around the kidney, and if the ureter is markedly dilated, its course may be traced from the hilus of the kidney to its entrance to the bladder, at which point the ureter will show little or no enlargement. In the mare, palpation of the ureters by way of the vagina instead of the rectum is advisable. Pressure on the kidneys often produces symptoms of pain. Changes in the ureters in the mare, as in the cow, consist mainly in dilation and excessive thickening of the walls. The accumulation of calcareous material in the exudate covering the mucous membrane of the bladder in the mare is a constant finding and represents the main marked differential pathology.

PATHOLOGY

Necropsy findings in typical cases having succumbed from the disease or having been destroyed in the advanced stages are highly characteristic. Emaciation of the cadaver is pronounced. The walls of the bladder are greatly thickened and the mucous membrane presents extensive folds that are edematous and hemorrhagic. A diphtheritic membrane containing an abundance of calcareous material is present in the case of the mare. The ureters are dilated up to 2 to 6 cm in diameter and contain large quantities of clotted blood, mucus and urine, the greatest dilation being present at the hilus. The walls are greatly thickened as the result of fibrosis. Both kidneys are usually enlarged and increased in weight, as the infection is seldom unilateral. The surfaces may present grayish opaque areas of various sizes, and normal lobulation is diminished in the bovine. Not infrequently numerous small abscesses of the cortical region cause adherence of the capsule. Upon removal of the capsule the abscesses rupture. The incised kidney of the bovine presents extensive changes of the calices and in the equine the pelvic portion of the organ is especially affected. The calices are filled with debris such as exists in the ureters. Petechial hemorrhages of the mucous surface and fibrosis of the walls are readily detected. Opaque, wedge-shaped-appearing areas are present in the papillae of certain cases, while in others large cavernous spaces filled with purulent material have replaced the papillary portion.

The chief histopathologic changes consist mainly of areas of marked leukocytic cellular infiltration with purulent centers. The

capillaries of the glomeruli are unchanged though bacteria may at times be found within Bowman's capsule. The epithelium of the tubules is destroyed and the tubules thus affected are greatly contracted if not entirely disintegrated. Other tubules contain massive accumulations of leukocytes and the interstitial tissue may be increased as a result of fibrosis.

Treatment

Though we have observed spontaneous recovery in one of our patients, a bovine, treatment either medicinal or surgical so far attempted by us has proved unsuccessful. A large percentage of the cases are not discovered until the disease has become extensive. Preventive measures consist of clean stalls for parturient and postparturient animals. In herds where the disease has appeared, catheterization of the bladder should be made in all females and the urine examined for the causative Corynebacterium. In a few instances we have found as high as two and three animals in the herd showing marked symptoms of the disease. The breeding of infected cows and mares should be discouraged, because of the danger of speading the infection.

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Discussion

Dr. T. H. Ferguson: Do I understand this is an ascending infection from the bladder? Is that the idea?

Dr. Boyd: I think that is right. I rather think that in most of the cases it is of an ascending type, and we have, to back that assumption up, the gross pathology and, in addition, the histopathology is rather indicative that that is the case in the embolic form of nephritis, which is hematogenous in nature. Then there are more changes in the glomeruli. That is rather characteristic in the embolic form, but in this type of disease, I think the infection is ascending in type in almost all cases.

Dr. Ward Giltner: Perhaps I did not follow your paper carefully, but is the disease reproduced experimentally?

Dr. Boyd: I do not think it is at all readily reproduced. I think it is a very difficult disease to reproduce.

DR. J. D. RAY: I should like to ask Dr. Boyd if he has ever had a history of some acute infection in these herds of cattle before he finds a chronic condition. I have seen some herds in which there were death losses with all the acute symptoms of nephritis, and in a few weeks you might have one or two of these cases of so-called pyelonephritis show up. In the acute cases the thing that would be outstanding, besides the acute symptoms, would be the fact that you apparently have some hemoglobin or blood in the urine. I was wondering if you have had that experience.

DR. BOYD: I might say, in answer to that question, that we have seen this disease frequently following an acute attack of mastitis or inflammatory conditions of the tubular portion of the reproductive organs. We have often seen that. At first we believed that diseases such as mastitis might be responsible for setting free certain organisms that later on might lodge in the kidney and start an inflammatory condition there and bring about a descending form or type of the disease, but this disease has been shown to occur in unbred heifers, and we have seen it so many times in animals that have not had any previous history of any acute infectious disease. It seems to me it just pops up here and there. I believe it would be contagious, but very slowly so. We found as many as three animals in one herd at one time. I do believe the disease is on the increase. We are certainly encountering a great many more cases than we formerly encountered, and probably we are better equipped to recognize them today than we were a few years ago. I had not seen any in horses prior to 1929.

DR. J. H. KRICHEL: I should like to ask Dr. Boyd if any work has been done in the way of treatment by using urethral catheterization and pelvic lavage with a suitable antiseptic.

DR. BOYD: I tried to catheterize the kidney to see what organisms I could get, but I did not succeed. I went to the urologist and discussed the way they do this in the human, and I got fully equipped with every kind of apparatus, but I did not succeed in catheterizing the kidney of the cow. The only time I have taken cultures directly from the kidney has been by the way of laparotomy, and then with a sterile hypodermic syringe. Aspirating from one of the lobules of the kidney, we have secured Corynebacteria in pure culture by way of laparotomy.

So far as the treatment is concerned, we have tried douching of the bladder, not in the cow, because I have not seen any cases until the disease was so extensive that I felt anything I would attempt would be useless. In the case of the mare, I have on numerous occasions practiced lavage of the bladder and thought I got fairly good results for a time, only later to find the gradual increasing thickness of the walls of the ureters.

DR. KRICHEL: I should like to ask another question. Have you ever tried intravenous medication?

DR. BOYD: Yes and no. I tried just a few times. I tried azamine, which is said to be directly eliminated by way of the kidney, but I presume it was not a fair trial as far as the drug is concerned, because the case was well marked. I have tried various remedies given by way of the mouth. We got up enough courage one year to do a nephrectomy, and we managed to pull through a ureterectomy, but our cow died ten days or two weeks later, and on autopsy we found that the right kidney was just as badly involved as the one we had removed. If one were going to do nephrectomy, one would have to be mighty certain as to the diagnosis.

DR. G. U. MARCHAND: What do you recommend?

DR. BOYD: I am recommending sanitation, especially care of the female just preceding and following parturition, and then if the disease appears in a herd, catheterize all the females in that herd and have

the urine examined for these Corynebacteria. We have been able to find a few in that manner. We advise against the breeding of animals affected with this disease. I have found many pregnant cows with this disease. Strange as it may seem, pregnancy in the bovine does not seem to aggravate this disease. Only when pregnancy is completed is the disease aggravated.

DR. DONALD W. BAKER: I had the bad luck to have charge of some cows back in 1923 and 1924, and I found that some of those cows died just before pregnancy. We had one cow that milked about 30,000 pounds and then died two or three weeks before she was to calve.

With respect to the etiology, I was interested because of a curious case that occurred in New York last year on sheep. Dr. Boyd spoke of this disease occurring in sheep, and I was going to ask him whether he thought the same organism produces the disease in cattle, horses, sheep and dogs, as has been reported. In these sheep it is interesting, because along in February or March the sheep suffered from acetonemia. I thought I was able to save some of them by giving them repeated small doses of pituitrin, removing the fetuses early. Some of these ewes recovered. An interesting thing about it was that, along in May or June, two sheep died. We were attracted to their condition because they became debilitated and they showed a frequent, scanty urination.

One of these sheep was brought to the postmortem room before it died. It was posted and an examination made. It was shown by bacteriological and pathological examinations, that this sheep had a pure infection. I wondered whether the acetonemia and the resultant poor condition of the animal following that might have allowed this to develop. I followed the history for about ten years, and I noticed that in a herd of about 110 head of cattle, they would have two or three cases develop every year.

DR. BOYD: Did the cases in the sheep occur in the female only?

DR. BAKER: Yes. Two of these ewes had acetonemia early in the year.

DR. BOYD: I suspect that they probably had the same form of nephritis, that is, pyelonephritis as we have observed it in cows. We have observed it in sheep only on one occasion in our work. Most of the trouble, as you know, in sheep occurs in the male, as far as nephritis is concerned, but we have found pyelonephritis, where we isolated Corynebacteria, on only one occasion in a ewe. I do not think it is at all common, and possibly depletion in these ewes as a result of the so-called pregnancy disease may have been a factor.

DR. M. G. FINCHER: I am very much interested in one phase of Dr. Boyd's paper. I wonder if this stallion was breeding mares that were infected with this disease.

DR. BOYD: I have every reason to believe that such was the case. He was present in a stud in which the infection had been found in a mare. Today we have isolated Corynebacteria from his urine and we can palpate his ureters quite easily. They are quite enlarged. I feel that he had served mares, or at least a mare, infected with this disease.

DR. FERGUSON: I have been bothered considerably with this disease in some pretty good herds and have had losses to the extent that owners were anxious to have their cattle checked to see if something could not be done to prevent it.

I wish to relate my experience in handling some of these cases or some herds where loss has occurred at the rate of two or three or four in a herd, always selecting valuable cows after parturition. In some of those herds, where the owner wished, we made a thorough examination of every cow shortly after parturition. We checked the ureters and the bladder and, as far as we could, the kidneys.

Our method of procedure was, first, to make the rectal examination to see if the ureters were enlarged. I talked to Dr. Jones one time and he assured me, just as did Dr. Boyd, that it was an ascending infection. If that is so, then in the early stages the bladder would be the organ affected. For that reason I thought it would be good judgment to wash out the bladder. I washed out the bladder with a soda solution, and then sometimes we injected a mild solution of formaldehyde. In every case we made it a practice to put all the animals we examined, whether they showed any symptoms or not, on formaldehyde, giving them about two or three ounces at day intervals.

I can say this much: It satisfies the owner or the herdsman, and I think I have seen some mild cases of it recover. Whether it was spontaneous or due to what little handling we gave them I do not know, but it is a satisfactory procedure. It satisfies the mind of your client and the herdsman that you are making an effort to find out more about the trouble and that you are also making an effort to do something about it.

Queer Things About Cats

Marguerite Warren-Burrill has written some interesting facts, in an article entitled "Queer Things About Cats," which appeared in a recent issue of *Our Dumb Animals.*

The writer states that white cats are invariably blind; that those on the Isle of Man and those of North Borneo are tailless, and that Egyptians must have considered cats as sacred animals because they mummified their remains. Mummified cats which have been unearthed have red hair, which color is believed to be due to the embalming fluids used.

The author states, also, that there are three times as many muscles in the tail of a cat as there are in the human hand and wrist. She has described the whiskers of a cat as feelers, and the conformation of the head as being a protection against concussion of the brain.

Horseshoes of Interest to Veterinarians

Under the above title, a booklet containing descriptions and illustrations of 164 types of shoes for remedial shoeing, used in lectures by Henry Asmus, professor of horseshoeing, New York State Veterinary College at Cornell University, Ithaca, N. Y., has been published by the author. Because of inquiries from both veterinarians and horse-owners who are interested in remedying defects of the foot and relieving numerous forms of lameness, the author has photographed and described the series of shoes used in class work in the Department of Horseshoeing over a period of 24 years. A copy may be obtained from the author for one dollar.

CONTROL OF PARASITES IN SHEEP*

By J. H. RIETZ, *Morgantown, W. Va.*

The control of parasites has become one of the major problems confronting sheep husbandmen throughout much of the sheep-raising area of North America. This problem is most acute in those areas where pastures are permanently fenced, and the sheep are confined to limited ranges. These fenced pasture areas are largely in the sections of the country where rainfall and moisture are adequate for crop production, moisture being one of the essentials for most, if not all, parasites for the development of that part of their life-cycle spent outside the host animal.

The so-called open-range country of the West is not entirely free from the parasite control problem. However, the hazards in the life-cycle of the parasite are considerably greater than in the small pasture and more humid areas.

RESEARCH AND EDUCATION

Specific research in connection with methods of control and treatment of internal parasites has received comparatively little attention until recent years. To be sure, some information was given to the world some 45 to 50 years ago, but most of this information was without adequate investigational background. Research leading to better control methods has been conducted by a few investigators during more recent years.

With the development of better methods of control, there is presented the problem of education of the sheep husbandmen. This phase of the work should be accomplished by the veterinary profession. The practitioner should become more active in this educational work than has been the case in the past. More research work and more educational work are needed before widespread control methods will be carried out.

Approximately ten years ago, the West Virginia Experiment Station became sufficiently interested in sheep parasites to begin investigations in a small way. The nature of this work has been treatment of the sheep plus proper feeding and management. Most sheep husbandmen and some veterinarians think of sheep parasites in terms of the stomach worm, but other parasites are quite in evidence.

In our work at the West Virginia Station, we encountered 16 species of parasites, exclusive of those found on the external sur-

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face of the body, and in one flock, purchased for experimental work, nearly all these species were found.

In table I are found the names of the parasites identified during our investigations.

TABLE I—*Parasites of sheep.*

SCIENTIFIC NAME	POPULAR NAME
Haemonchus contortus	Stomach worm
Oesophagostomum columbianum	Nodular worm
Oesophagostomum venulosum	Nodular worm
Ostertagia sp.	.....
Cooperia sp.	.....
Nematodirus sp.	.....
Bunostomum trigonocephalum	Hookworm
Trichuris ovis	Whipworm
Chabertia ovina	.....
Moniezia expansa	Tapeworm
Moniezia alba	Tapeworm
Dictyocaulus filaria	Lungworm
Synthetocaulus rufescens	Lungworm
Eimeria faurei	Coccidia
Cysticercus tenuicollis	Tapeworm cyst
Oestrus ovis	Grub

The identification of the species of parasites represented in the flocks was made from autopsy of untreated control animals, treated animals slaughtered, and animals dead from other causes.

Fecal examinations were made of all sheep to determine the number of nematode ova per gram of feces and to determine the presence of ova or segments of cestodes. These examinations were made before treatment was begun and at regular intervals during the treating process and always at the termination of the treatments. A total of 162 sheep, exclusive of untreated controls, have been used in the series of treatments. These sheep were handled in lots of 20 to 30 individuals. All treatments were administered at intervals of 21 days.

The longest time that any one lot received treatment was 63 weeks and the shortest time was 15 weeks. Individual animals were slaughtered for autopsy from each lot at much shorter intervals. The drugs used were:

Copper sulfate 1 per cent.
Copper sulfate 1½ per cent.
Mixture of copper sulfate 1½ per cent and nicotine sulfate 1½ per cent.
Nicotine sulfate 1½ per cent.
Tetrachlorethylene.
Colloidal iodine.
Calcium polysulfite.

Experimental Treatments

Calcium polysulfite: Ten sheep were treated with calcium polysulfite. The sheep were prepared for treatment by starving them for 24 hours. The drug was given in 1 per cent to 12 per cent solutions but the dosage was varied from 12 ounces to 1 ounce. This preparation was quite irritating to the mucous membranes, was a poor anthelmintic and caused the death of most of this group of sheep.

Tetrachlorethylene: Four sheep were prepared for treatment by starving them for 24 hours, and 5 cc of tetrachlorethylene was administered. The nematode ova count was not reduced so rapidly as with some other preparations and the cestode infestation was not relieved.

Nicotine sulfate: Four sheep were prepared for treatment by starving them for 24 hours, and 3 ounces of a 1½ per cent solution of nicotine sulfate was administered. This preparation nauseated most of the sheep but was quite effective in the reduction of nematode ova and cestode infestation.

Colloidal iodine (Merck): Four sheep were prepared for treatment by starving them for 24 hours, and 20 cc of colloidal iodine was administered. In our hands this preparation was ineffective in the removal of either nematode or cestode infestation.

Copper sulfate: Fourteen sheep were prepared for treatment by starving them for 24 hours, and an average of 8.15 treatments with 3 ounces each of a 1 per cent solution of copper sulfate were administered. This resulted in a 71.7 per cent reduction in the nematode ova during the period of treatment. Cestode infestation apparently was not affected.

Eleven sheep were prepared for treatment by starving them for 24 hours and an average of 14 treatments with 4½ ounces each of a 1 per cent solution of copper sulfate were administered. This resulted in a 50.8 per cent reduction in nematode ova. Cestode infestation apparently was not affected.

Sixteen sheep, consisting of adults and yearlings, without preparation for treatment by starving, were given an average of 7.45 treatments with 4½ ounces each of a 1 per cent solution of copper sulfate. This resulted in a reduction of 81.1 per cent in the nematode ova. Cestode infestation apparently was not affected.

Eleven sheep that had not been prepared for treatment by starving were given an average of 14.8 treatments with 3 ounces each of a 1½ per cent solution of copper sulfate. This resulted in a reduction of 76 per cent in nematode ova. The cestodes were partially controlled.

Forty-six sheep were prepared for treatment by starving them for 24 hours and an average of 7.1 treatments with 3 ounces each of a 1½ per cent solution of copper sulfate were administered. This resulted in a reduction of 95.6 per cent in the nematode ova. The cestode infestation was completely controlled. In one of the lots receiving this treatment the nematode ova were reduced 98.7 per cent during the period of treatment.

Eleven sheep were prepared for treatment by starving them for 24 hours and an average of seven treatments with 3 ounces each of a mixture of equal parts of a 1½ per cent copper sulfate solution and a 1½ per cent nicotine sulfate solution were administered. This resulted in a reduction of 98.2 per cent in nematode ova. Cestode infestation was entirely controlled in this group.

The untreated control animals in all lots showed an increase in parasitic infestation and were autopsied when moribund.

Starving

Starving increased the effectiveness of the treatments approximately 20 per cent in the reduction of nematode ova.

An effort was made to determine the reason for the more effective results following starving of the animals. Slaughter animals starved and not starved were drenched in equal numbers with a dye solution just before slaughter. Examination of the digestive tract of these animals showed that part or all of the dye solution had entered the abomasum and duodenum in the starved group, while in the group not starved, many animals showed that none of the dye had entered the abomasum or duodenum.

Starving the sheep before drenching in some way affects the passage of the drench solution to the abomasum and intestines where it comes in contact with the parasites, while in sheep not starved, much more of the drench solution is lost in the rumen and other divisions of the stomach.

Mass Feeding

Drenching sheep at intervals of 21 days throughout the year is considered a difficult task by some sheep husbandmen, consequently an effort was made to mass feed copper sulfate in a salt mixture.

Twenty sheep that were practically free from parasites were placed in what was considered a heavily infested pasture and allowed access to a mixture of copper sulfate and salt, 1:30. The consumption of this mixture was ½ pound per month. Before the end of one year, three sheep of this group had died of cop-

per sulfate poisoning; also the nematode ova count in the feces had materially increased, although the general condition of the flock had not been visibly reduced.

Because of the loss of three sheep from copper sulfate poisoning, the copper sulfate-salt mixture was changed from 1:30 to 1:50 at the end of one year. One additional sheep died from copper sulfate poisoning just 21 days after the change from the 1:30 to the 1:50 mixture of copper sulfate and salt.

Three months following the change from 1:30 to 1:50, the ova count both of nematodes and cestodes had materially increased and the condition of the sheep had decreased, so that mass feeding was discontinued.

The sheep in this flock were now divided into two lots. In one, each sheep received 3 ounces of a 1½ per cent solution of copper sulfate, and in the other lot, each sheep received 3 ounces of a mixture of equal parts of copper sulfate and nicotine sulfate. The interval between treatments was 21 days. After five months of treatment, there was a reduction of 98.7 per cent and 98.3 per cent, respectively, in nematode ova in the two lots and the cestode infestation was eliminated entirely in both lots.

The West Virginia Experiment Station flock, numbering about 75 head of purebred sheep, has been treated with the 1½ per cent solution of copper sulfate every 21 days for more than seven years. The dosage for adults and yearlings has been 3 ounces. Lambs, eight to ten weeks of age, were given 1 ounce. No ill effects have resulted from the administration of the copper sulfate and the flock is in excellent condition.

A critical test of the seven solutions, drugs and combinations, indicates that a 1½ per cent solution of copper sulfate in dosages of 1 ounce and up for lambs and 3 ounces for adults, administered at intervals of three weeks throughout the year, will effectively control the gastro-intestinal parasites enumerated in this report. Dieting for 24 hours before treatment is essential. Pregnant ewes are not dieted during the lambing period and for two treating periods following lambing.

LUNGWORMS

Lungworm infestation has received some attention in West Virginia. The routine method of treatment has been anesthesia with chloroform. This has given fair results but is not unattended with danger of losses from pneumonia following the anesthesia.

Approximately 250 head of sheep, in which lungworms were definitely diagnosed, have been treated with an oily solution of pyrethrum. The pyrethrum solution was manufactured from 3

pounds of pyrethrum flowers extracted in 1 gallon of oil. The pyrethrum solution was injected directly into the trachea. The dosage ranged from 3 cc to 6 cc. However, the results were not different in those receiving the larger dosage; consequently, only a few sheep received the 6-cc dose. One treatment was sufficient for 70 to 75 per cent of the sheep treated, and only 3 to 5 per cent required a third treatment before larvae could not be demonstrated in the feces.

No losses from pneumonia resulted following the use of this pyrethrum solution.

Feeding

In all this work of testing drugs and methods of treatment, feeding is markedly reflected in the results obtained.

When the feed is of good quality and quantity, the count of ova always decreased more rapidly and remained at a lower level than in times of a shortage of feed or feeding a poor quality of feed.

Feeding will not compensate for systematic treatment nor will systematic treatment compensate for poor feeding.

Maximum results in the control of sheep parasites are reflected in good feeding and management plus systematic treatment.

Discussion

Dr. C. W. Fogle: In computing the nicotine sulfate, do you make a solution of 1½ per cent of the liquid nicotine sulfate as you buy it or absolute nicotine sulfate? As I understand, most of it is only 40 per cent absolute nicotine sulfate.

Dr. Rietz: It is a 1½ per cent solution of the 40 per cent solution.

Dr. Donald W. Baker: I believe that practitioners are becoming better acquainted with the treatments that have been developed recently for controlling intestinal and lung parasites in sheep. This past year, I have had an opportunity to meet a considerable number of sheep men. In New York State I attended meetings in 13 counties; sometimes there were two or three meetings in a county, and I estimate that I met more than 1,000 good sheep men, some of them feeders, but most of them breeders.

In this group I found that the men were very much interested in parasitisms. They realized that parasite control was more important than any other single factor in deriving a profit from their sheep business. My experiences were very much as has been described.

We have a great variety of parasites. I have listed twelve that I found this past year. We found some unusual cases. In one instance, a man had bought 75 ewes from Montana. They were beginning to die, and this man, a college graduate, who was versed in handling sheep, was very much disturbed because he had treated them for parasites without satisfactory results. They continued to die. On postmortem, I found that one animal had practically a pure infestation of what we called the Nematodirus worms, a very unusual circumstance.

We have the lungworm problem and as yet have not been able to work out any very good method for controlling it. We have both kinds of lungworms, the large one found in the bronchioles, and the smaller one found in the lung tissue. I think a good many of them eliminate the large lungworm, but we find some difficulty in controlling the other one.

In New York State, the common stomach worm is not a problem in the better sheep flocks because the men know how to control it. They are using copper sulfate solution. We have gone through the period of the popularity of various drugs, including Fowler's solution and others mentioned, but the farmers are finding that copper sulfate solution, 1½ per cent, or 1 per cent, and varying the dose, is more effective than anything else. This does not control the small intestinal worms. As yet we have no method of control.

The other point mentioned was that feeding is very important. On a farm where proper feeding practices are carried out, the animals are able to withstand the heavy infestations of parasites much better than on the places where the feed is poor in quality and small in amount.

There is one other point which I think might be of interest to you practitioners: A few years ago, some of the large biological houses and men supplying anthelmintics started the sale of copper sulfate tablets, small tablets which are easy to use—that is, the farmers think they are—but I found that they are practically ineffective. In demonstrating the use of the various anthelmintics to the students, I have found that upon slaughtering sheep up to 30 minutes after the administration of the tablets, they are usually to be found in the rumen. They have not passed back to either the abomasum or the small intestine where they might be of some value.

The matter of starving before the administration of an anthelmintic, I think, is very important. Many of the sheep men in New York State regularly starve their sheep for 24 hours before they administer the anthelmintic. They find the copper sulfate and nicotine sulfate solution, as previously described, very effective for the tapeworms.

There is one question in my mind: Over a period of time, a good many sheep eliminate their tapeworms without any anthelmintic treatment, and I have always questioned the usefulness of any drug that I have tried in removing this particular type of tapeworm.

Probably one unusual parasite which very few men have seen was brought to my attention not long ago. The liver of a sheep which had been secured from a slaughterhouse was presented to the laboratory, and upon examination was found to be practically filled with ascarids. Unfortunately, I was unable to get these worms out of the bile ducts in very good shape. In case any of you encounter such a condition in a slaughtered sheep, I should like to get some more specimens.

DR. M. J. JONES: How long do you diet these sheep after the treatment?

DR. RIETZ: We treat them immediately. As soon as we are through treating, we turn them loose.

DR. E. C. W. SCHUBEL: Then you would say it is perfectly safe to give 1½ per cent of nicotine sulfate and 1½ per cent of the copper sulfate solution instead of what we have been giving in the past?

DR. RIETZ: Yes. I presume in West Virginia we treated 100,000 head of sheep with that 1½ per cent solution of copper sulfate. I do not know how many thousand have been treated with the combination, the 1½ per cent solution of nicotine sulfate and 1½ per cent of copper sulfate in equal quantities. There was no greater mortality than from the ordinary accidents which will happen.

This material is administered almost universally in West Virginia with a dose-syringe. I might say one other word as to an experience we have had in the administration of any liquids to sheep. We have put them in the ordinary recumbent position and drenched them with a dye solution and then slaughtered them within the next hour. We have yet to find the first sheep in which that solution did not enter the rumen. So you might just as well not treat a sheep at all as to

give it its dose in a lying position. They must be standing or it will not pass back into the abomasum.

DR. T. H. FERGUSON: In treating those large flocks of sheep, the item of speed must enter into it. Do you find that you make as good speed with the dose-syringe as with the stomach-tube?

DR. RIETZ: We have not used the stomach-tube at all. We have used the dose-syringe entirely.

DR. FERGUSON: My experience has been limited to small flocks, but the item of time is always pressing us. My system for administering that solution, or any other solution to sheep, is to have enough assistants to hold them and to have the operator pass the stomach-tube with a funnel on the end of it. Then an assistant pours in the solution. We can go through a flock of sheep very fast that way and not make any mistakes. They get the sheep up and hold them in an upright position, place one hand on the upper jaw and pass the stomach-tube with the other. Then the solution is poured in. You can go very fast that way.

DR. RIETZ: Last Monday we treated our experiment flock, which consists of 60 head at the present time, and it took me just 45 minutes with one assistant to treat the 60 head. I timed myself.

DR. FERGUSON: I think that is as fast as anybody can step.

DR. I. B. BOUGHTON: We have what are known as sheep dopers in Texas. They are quack ranch hands. It is a poor sheep doper who cannot dope 400 sheep an hour.

DR. FERGUSON: I do not know much about the sheep dopers, the common run of them, but I have had this experience with Thoroughbred horses: I have been called in to a band of horses that were wormed in the past season and they showed me the capsules they picked up. I should like to see somebody slip the capsules into some of these hot-headed Thoroughbreds. But when you get the stomach-tube down, you get your medicine down.

DR. BOUGHTON: We have the same trouble. They do it as fast as 400 an hour.

Animal Life in Death Valley

When reading about Death Valley, California, which is 270 feet below sea level, the inference is that no animal life can exist there. This valley has an abundant plant and animal life. Sheep that are closely related to the Rocky Mountain variety, whose natural habitat is high above the timber line, live entirely on the plants of the desert. According to Jasper B. Sinclair, writing in *Our Dumb Animals*, it is not known when their ancestors migrated from the chilly mountain peaks to this sun-scorched valley. These sheep may be seen flocking around far-scattered watering places.

Two other animals also may be found in this valley. One is the tiny pocket-mouse. The other is the kangaroo rat which, like its big brothers, the kangaroo and the wallaby, can travel with equal facility on two or four legs. It is believed that both these rodents exist chiefly on plant seeds and never touch water.

THE USE OF ACIDOPHILUS MILK IN THE TREATMENT OF DYSENTERY OF YOUNG ANIMALS*

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INTRODUCTION

Dysentery takes a heavy toll of young animals every year; this is especially true in lambs and calves. The losses in lambs is especially heavy during severe winters on ranches where sheds are used in the lambing operations. In calves the trouble does not appear to be so seasonal as in lambs but in districts where calving occurs at a time when advantage may be taken of some special forage crop, losses seem to be greater than in other districts.

The causes of this disease have been quite thoroughly investigated by numerous workers, namely, Jensen,[1] Carré, Christiansen, Smith,[2] Heller, Tunnicliff, and others. These workers have established as the cause various aerobic and anaerobic bacterial invaders of the intestinal tract.

Having in mind the success of attempts[3] to change the intestinal flora of human beings and monkeys by using culture milk, it was thought that certain fermented milks might be useful in relieving enteritis in young animals. Since one of these fermented milks was made by using an organism found to be an inhabitant of the bowel of human beings and domestic animals, this organism was chosen as the one most likely to prove successful.

PRODUCTION OF ACIDOPHILUS MILK

Three different strains of organisms were used in this work. Two strains, S and P, freshly isolated from human intestines, were obtained from Dr. George Valley, of the Brady Laboratory, in July, 1935. The other, O, was obtained from the Bacteriology Department, Oregon State Agricultural College. This strain had been cultivated for several years in cows' milk. Its original source was unknown.

Strains S and P developed typical rough colonies when cultured on tomato-juice agar plates. Strain O developed round, smooth colonies when grown on the same medium. All strains readily reduced litmus, but milk was coagulated somewhat more rapidly

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by strains S and P than by O. The cultures were occasionally plated on tomato-juice agar and examined microscopically to insure purity.

The milk used consisted of skim milk obtained from a herd of mixed breeds. After being brought to room temperature, it was placed in 3-litre Erlenmeyer flasks, in 2-litre amounts. It was then placed in an autoclave and sterilized at 15 pounds extra pressure for 40 minutes. Milk so treated possessed a rich cream color and a distinct "canned milk" flavor.

It was found that these properties were a practical index of sterility. Milk not sufficiently heated to cause a distinct change in color was frequently not sterile. Slight over-heating causes a coagulation of the casein, which makes the milk undesirable for use. As *Lactobacillus acidophilus* offers very poor competition to some of the other organisms found in the milk, proper sterilization is very important.

After cooling to 37° C., each flask was inoculated with approximately 30 cc of fresh culture, and incubated at 37° C. for about 18 hours, or until coagulated. Coagulation is attended with sufficient lactic-acid production to check the growth of most contaminating organisms. It is not necessary, therefore, to handle coagulated milk in a bacteriologically sterile manner.

When kept at room temperature, the cultured milk slowly developed a slightly firmer curd and became more acid. Although there was little change in appearance, the milk was discarded if not used within 15 days after being removed from the incubator.

For distribution, the milk was placed in 1-gallon glass containers and shipped by express, with instructions to protect it from freezing and temperatures exceeding 80° F.

ANIMALS TREATED

Lambs: The first lambs[4] treated were from registered ewes, dropped in a shed mostly under apparently good sanitary conditions. Though a good many remedies had been used, all lambs developing the disease had died. Fourteen lambs were showing symptoms. These lambs were from two to five days old and in various stages of the disease. Marked improvement followed the use of two ounces of milk three times daily. On this ranch, during the lambing season, a total of 46 lambs were treated, including the 14 sick on the first day. Of this number, 22 lived and 24 died. No changes in management were recommended.

The following year, milk was shipped to eastern Oregon to try on range lambs; while the dysentery was not severe that

year, good results were reported in the cases where the milk was used.

This year (1936) proved a severe winter and dysentery began causing heavy losses among the early lambs. In sub-zero weather the treatment received its most critical test. Over 300 gallons of milk were shipped to the counties where losses were occurring, and approximately 15,000 lambs were treated. Most of these lambs were given a 1-ounce treatment before being allowed to nurse the ewe. Then, if scours developed, 2-ounce doses were given until the lamb either recovered or died. The milk was used on 15 different ranches and all reported favorable results.

In some instances the first milk used did not seem to give desired results; investigation proved that this was because the milk was not given proper care during shipment or after reaching the ranch.

Calves: The first opportunity to try the milk on calves[5] occurred in July, 1935, at a dairy operated near the Station by a dairyman above the average in intelligence. On this ranch the losses had been 50 per cent up to the time treatment began. The scours in calves of this herd was quite typical, the disease appearing when the calves were only a day or two old and causing death in from 24 to 72 hours after symptoms appeared. These calves were kept in dark, insanitary pens, but apparently this was not a factor, as calves developed the disease when kept in a clean part of the barn after being dropped in a stall that was fairly clean for a maternity stall.

When the owner called for assistance with his problem, the information on the use of milk in lambs had just been published and considerable milk was on hand. A total of 26 calves were born in the herd during the year. Before treatment began, eight calves were born that did not scour; four scoured, two of which died. Following the beginning of treatment, 14 calves were dropped. Eleven scoured. Two bull calves not treated died and nine recovered following treatment. Three calves did not develop symptoms when treated before being placed in pens.

In another herd the treatment failed to save bull calves after symptoms developed, but proved 100 per cent efficient in preventing losses in heifer calves when milk was given a few hours after birth. It did not in all of these cases invariably prevent the development of the symptoms, but losses did not occur. In this particular herd the bull calves were used as checks against the heifers, as they were only grade calves without value. Eventually the virulency of this infection died down until even the

bull calves failed to develop the trouble. Heifer calves continued to be treated, however, with the idea of prevention in mind.

In a purebred herd eleven calves began scouring at about the same time and, even with the care of two veterinarians, within four days five calves had died and one was so nearly dead no hope was held for it. Milk was given the remaining six, and the five not down were very much improved the next day. One stopped scouring entirely and continued well, while four kept on scouring but were apparently not sick. A commercial acidophilus milk was then used, but without very much effect. At this time four more calves were born, and in spite of being treated with the commercial product, developed the disease. With nine calves scouring, a supply of culture milk was obtained from the Experiment Station and in four or five days all calves had recovered. Here again the disease seemed to lose its virulency, since no new cases developed in the calves born later.

A certain type of calf scours causes heavy losses in our range calves each year. These calves are dropped usually in March when the cows are fed mostly grass hay. For this reason it has been thought the trouble was due to a vitamin A deficiency. The use of cod-liver oil seemed to give fair results in treating the disease. It was with little hope of success that the acidophilus culture milk was tried in these cases. After the use of some 30 gallons, it was apparent that desirable results were not to be obtained.

Colts: The milk was used in two cases of scours in colts two or three weeks of age. In the one case the colt did not appear sick in any way except that the bowels were very loose. This animal was given 6 ounces of the cultured milk, and in 24 hours the feces appeared normal. It was not so easy to give the milk to the colts as it was to the lambs and calves, but in this region scours in foals has never caused much loss.

Pups: No experimental work has been done with the use of this milk in puppies, but Dr. G. H. Huthman, of the Huthman Hospital, in Portland, Oregon, reports very satisfactory results from its use in both ordinary dysentery in small pets and dysentery accompanying distemper. Records were not kept on the number of pups receiving milk.

Pigs: In a personal communication to the authors, A. H. Quin, Jr., of Des Moines, Iowa, stated that acidophilus milk had given good results with pigs several years ago, but that the results had not been published. Dysentery in pigs is not a problem in this state; hence very probably if cultured milk is used to treat this disease of pigs it will be used where losses occur.

Discussion

The use of acidophilus culture milk is both theoretically correct and entirely practical. It is not claimed that it will prevent or cure all cases of dysentery; but plenty of evidence is at hand to show that very favorable results are obtained when properly cultured milk is properly handled and used in cases of ordinary dysentery in lambs and calves.

An attempt is being made to isolate strains of *Lactobacillus acidolphilus* from the intestines of animals in which the culture milk is to be used. It is thought that such strains might be more capable of establishing themselves when sown than strains isolated from the human intestine.

The question is often asked by the stockman, "Can this product be made on the average farm?" Experience of the authors convinces them that the milk can be successfully produced only under laboratory conditions.

In human medicine it has been found advisable to establish new cultures every three or four months. Some of our unfavorable results were with milk produced with a strain that had been isolated for six years. Some bacteriologists have apparently failed to understand the principle underlying the therapeutics of acidophilus milk. This principle depends on the number of viable organisms in the milk, the milk merely acting as a medium.

Some difficulty has been experienced in having stock-owners realize the difference between acidophilus milk and milk soured by other lactic-acid producing organisms. It has been quite definitely proved that it is not the acid that is responsible for the results obtained.

Summary

Lambs, calves, colts and pups have been successfully treated for dysentery by the use of milk soured by *Lactobacillus acidophilus*. The milk, to be effective, must be properly made and cared for.

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DISEASES OF THE PROSTATE GLAND IN THE DOG*

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Diseases of the prostate gland, as observed in man, are in lower animals almost entirely restricted to the dog. Hypertrophy of this organ is common in the dog and not infrequently is the cause of symptoms. Most of the textbooks on diseases of lower animals mention various diseases of the prostate gland but do not stress their importance. It has been my observation that diseases of the prostate gland are of great importance as regards the health and longevity of male dogs. Because of that, it was thought that the subject merits further consideration.

The frequent occurrence of disease of the prostate gland in the dog may be attributable to its anatomic and functional development. The prostate gland of the dog is relatively large, dense in structure, and globular in shape. It surrounds the neck of the bladder and urethra at their junction. A median furrow divides it into two lateral lobes, but lobules of prostatic tissue are also found in the wall of the urethra for a short distance distal to the prostate gland. The median lobe is poorly developed or absent. The urethral crest is quite prominent (fig. 1). The capsule and stroma contain a large quantity of smooth muscle which, when it contracts, causes the gland to empty its secretion into the urethra through numerous ducts.

Sisson[1] mentioned that there is great variation in the size of the prostate gland of the dog, but did not state the size of the average normal gland. In 1932, I[2] said that any prostate gland weighing more than 10 gm probably was abnormal. However, since then, Bollman and I[3] have found that this standard of size is not adequate. A large dog should and does have a larger normal prostate gland than does a small dog. It was learned that in dogs there is a constant relationship between the weight of the prostate gland and the weight of the body until five years of age. We found that there is 0.1 to 0.7 gm of prostate gland for each kilogram of body weight (fig. 2), the average weight being approximately 0.4 gm of prostate for each kilogram of body weight. After the age of five years, this relationship varies markedly. This agrees with the observations of L. W. Smith.[4]

Wiggers[5] said that the prostate is a gland of external secretion and that the hypothesis that it elaborates hormones which

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promote spermatogenesis and increases sexuality date from the observations of Scherlack and Pares. Subsequent investigations tend to indicate that the spermatorrhea which they noted following subcutaneous injections of prostatic extracts into laboratory animals is attributable to increased secretion of fluid which results from hyperemia of the seminiferous tubules. This merely flushes the spermatozoa from the tubules and is actually followed by aspermatogenesis. Wiggers mentioned that removal of the

FIG. 1. Cross section of an essentially normal prostate gland obtained from a dog. Note the absence of a glandular median lobe (x 6½).

prostate gland before puberty does not appear to affect the development of sexual characters. When the prostate gland of the adult is removed, it does not affect sexual desire but appears to decrease fertility. Smith[6] said that the secretions of the prostate gland excite energetic movements in the spermatozoa. He also referred to the investigations of Iwanow, who discovered that the prostate gland in the dog furnishes the third ejaculation.

Bell[7] said that hypertrophy of the prostate gland of man is by far the most common disease of this organ and that in about 8 per cent of men who are past 60 years of age the condition is sufficiently pronounced to cause symptoms of obstruction. He further said that prostatitis is secondary and is not the cause

of hypertrophy. Prostatic hypertrophy is considered a neoplastic process consisting of multiple fibroadenomas with fibromyomas. Ten to 15 per cent of enlarged prostates in man are carcinomatous, a few are tubercular, and in a small number of cases the enlargement is caused by pyogenic infection.

Hypertrophy and other lesions of the prostate gland of the dog have been observed by Goodpasture and Wislocki,[8] Goodpasture,[9] L. W. Smith,[4] Nieberle and Cohrs,[10] Lund,[11] Law,[12] Dollar,[13] Hobday,[14] French,[15] Williams and Williams,[16] Müller,[17] Wooldridge[18] and others. In my observations on two large series of prostate glands obtained from dogs of all ages, approximately 60 per cent of these glands could be classified as histologically ab-

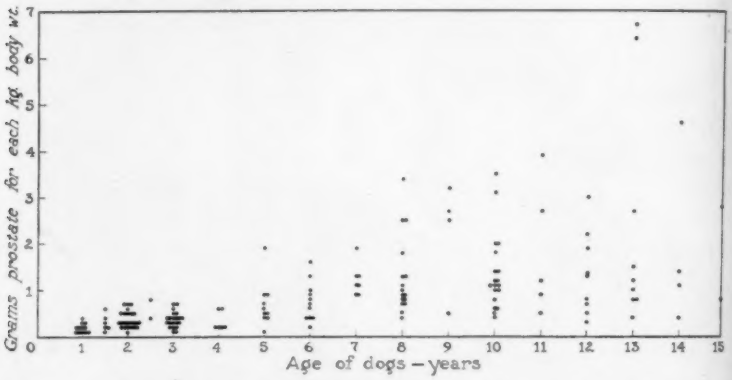

FIG. 2. Variation in the size of the prostate gland in dogs after the age of five years.

normal. Benign hypertrophy was by far the most common disease noted. Hypertrophy and cystic degeneration were frequently encountered in the prostate glands of aged dogs. Some glands were greatly enlarged. The largest gland weighed 260 gm and contained huge cysts (figs. 3 and 4). Suppurative prostatitis was not infrequent in aged house-trained dogs. In some, solitary and multiple abscesses could be seen macroscopically, while in others, collections of leukocytes were observed microscopically (fig. 5). Abscesses of the prostate gland occasionally attain huge size. The largest observed by me was 11 by 8 by 5.5 cm. It caused marked compression of the urinary bladder which resulted in the death of the dog. When the abscesses are multiple they generally are small in size and cause irregular enlargement of the prostate. Adhesions to the other organs are frequent. Chronic prostatitis, manifested by areas of lymphocytic infiltration and fibrosis, was not infrequently observed

(fig. 6). Tuberculous prostatitis has not been observed in the dog. Law[12] mentioned that Goubaux said that cancer of the prostate is common in the dog. Other authors mention the occurrence of sarcoma and carcinoma in the prostate gland of the dog, but I have not observed similar lesions.

Nieberle and Cohrs[10] said that in hypertrophy of the prostate gland of the dog the glandular tissue occurs in nodules which are surrounded by a web of connective tissue so that multiple gland-like structures are observed in cross sections of the entire

FIG. 3. Cystic degeneration of the prostate gland sectioned across through the gland and urinary bladder.

prostate gland. The urethra may be compressed and partially obstructed so that compensatory muscular hypertrophy of the bladder may result. I have noted that one cannot estimate the degree of obstruction present in the urethra by knowing the size of the prostate (fig. 7). A large gland may cause no apparent obstruction, whereas in a small one the obstruction may be marked.

A review of the literature tends to reveal that there are various causes of disease of the prostate gland. Lund[11] was of the opinion that hypertrophy of the prostate gland is primary and that glandular, interstitial and muscular hypertrophy are all vari-

ous stages of the same process. House life, overfeeding, retention of urine and feces, proctitis, urethritis, cystitis, excessive sexual excitement, urinary irritants, infections, and trophic derangement when function declines were mentioned by Law[12] as various causes of prostatitis and hypertrophy of the prostate gland in the dog. However, none of these factors have been proved to be of etiologic significance. Müller[17] said that a number of causes lead to inflammatory processes in the prostate gland and produce

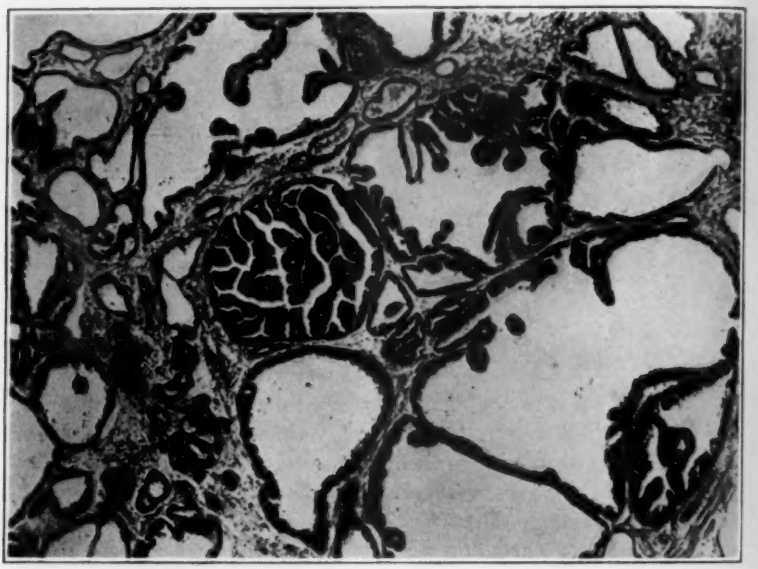

FIG. 4. Cystadenomatous hypertrophy of prostate gland (x 50).

hypertrophy or neoplasia of this organ. He mentioned that prostatitis is observed in stud dogs that have served a large number of bitches within a short space of time. French mentioned that acute prostatitis is uncommon but that the chronic type is not infrequent. He suggested that it is caused by microbic invasion, usually through the urinary tract. Nieberle and Cohrs[10] said that infectious agents which cause prostatitis arise from the kidneys, seminal vesicles and hematogenously. When the condition is hematogenous in origin, the infection generally is metastatic from some other primary focus. Nieberle and Cohrs said that the cause of prostatic hypertrophy is still unexplained.

The influence of certain specific hormones on the growth and development of the prostate gland has been observed by many

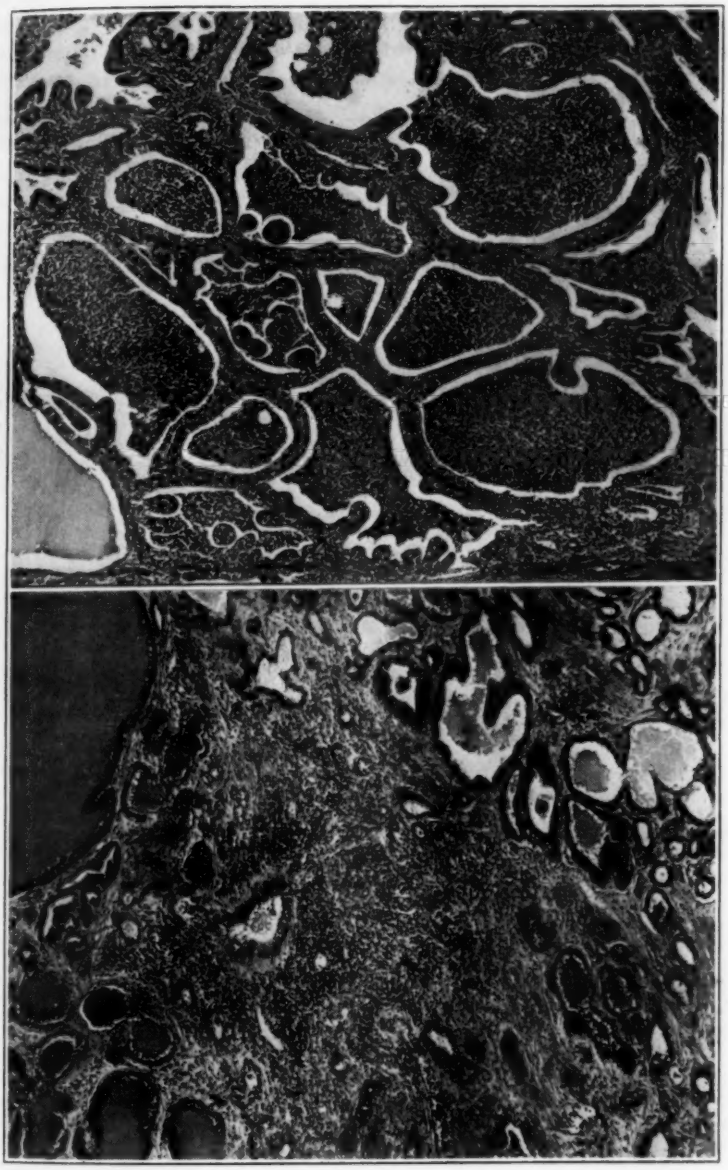

FIG. 5 (above). Purulent prostatitis (x 50).
FIG. 6 (below). Chronic prostatitis showing cellular infiltration (x 60).

investigators. Lower and Johnston,[19] in 1931, reviewed the work of Fishera; Haitai; Smith and Engle; Smith and MacDowell and of Evans and Simpson as regards the interrelationship of the pituitary body, thyroid gland, suprarenal glands and sex glands. They at that time reported results of their own investigations. According to their researches, an excess of anterior pituitary hormone has a stimulating effect on the gonads and prostate gland of normal animals. The prostate gland, being an accessory organ, receives its stimulation through the testes, because if the testes are removed, atrophy of the prostate gland and seminal vesicles results. The maintenance of a normal testis

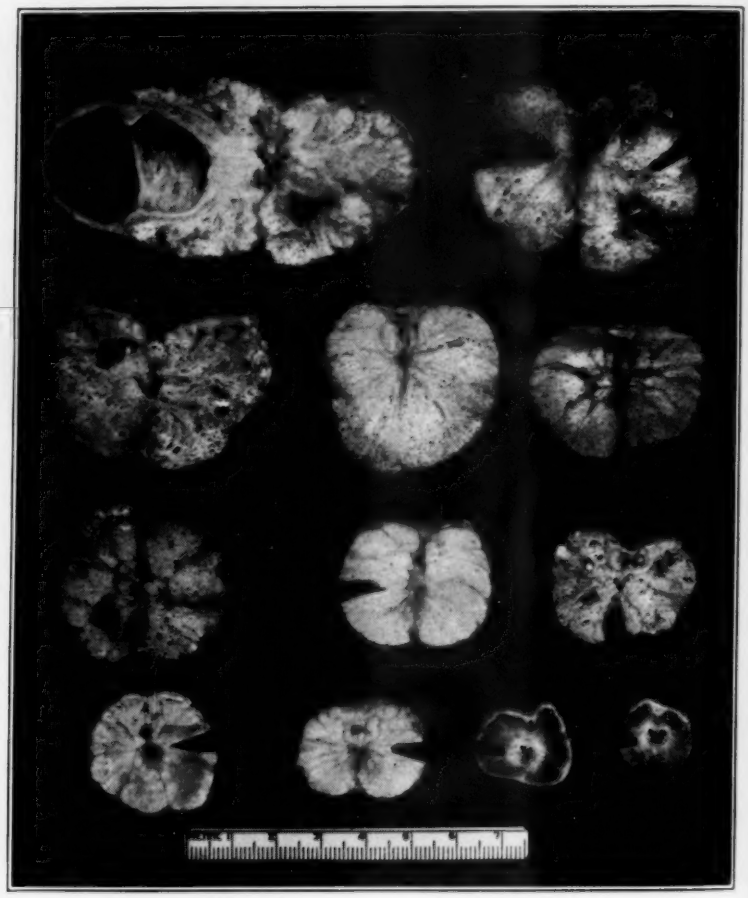

FIG. 7. Cross section of prostate glands obtained from dogs of various ages. Note various degrees of hypertrophy and urethral obstruction. The two small glands are essentially normal.

is dependent on an intact anterior pituitary body; the maintenance of a normal prostate gland is dependent on testes which are capable of producing adequate hormone, but not an excess. Testes lacking in reproductive function, those in which the tubules have undergone marked degeneration for atrophy, instead of causing atrophy of the prostate gland have a tendency to cause hyperplasia.

The tubular cells of the testes play no part in the production of the male hormone but they are credited with neutralizing or absorbing the excess of gonad-stimulating hormone of the pituitary body and thus prevent hypertrophy of the prostate gland. Therefore, atrophy of the tubules, as occurs in old age, in cryptorchids, and following exposure to roentgen rays, is followed by hypertrophy of the prostate gland. Lower,[20] in subsequent experiments, found that the anterior pituitary body stimulates the interstitial cells of the testes and thought that the tubules of the testes elaborate a hormone that inhibits the action of the pituitary body. The interstitial cells of the testes elaborate the male sex hormone that causes normal development of the prostate gland and seminal vesicles. The excess of male sex hormone is eliminated in the urine. It has been shown that the male hormone obtained from the urine of normal males, when injected into animals, is capable of producing prostatic enlargement.

I have observed that hypertrophy and other diseases of the prostate gland are more frequent in house-trained dogs than in dogs maintained under other environmental conditions. The etiology of this, of course, is not known.

The symptoms of disease of the prostate gland vary. The gland may be markedly diseased and yet not cause noticeable symptoms; much depends on the type and degree of disease present (fig. 5). Müller[17] mentioned that prostatitis may cause a dog to walk with the back arched and a stiff, stilted gait. There is evidence of pain when either urine or feces is passed. Frequently, a dog will make several attempts before accomplishing these acts. Because of the pain associated with the act of defecation, constipation is frequent. Loss of appetite, fever and depression are frequently noted. Palpation of the prostate gland through the abdominal wall and through the rectum may reveal an enlarged, firm, hot and painful gland. If suppurative prostatitis is present, pus may be expressed into the urethra in sufficient quantity so that it will exude. It has been my observation that large abscesses generally are encapsulated. Fluctuation sometimes can be noted on palpation. Symptoms similar to

those described have been observed by Hobday,[14] French[15] and others.

In addition to these symptoms, I have observed that hypertrophy of the prostate gland and prostatitis frequently cause marked weakness of the hind legs. This may be so severe that the animals lose control of their hind legs and they will drag themselves as if paralyzed. Constipation and retention of urine generally are associated with this symptom. If the urethra is incompletely obstructed, the bladder may be greatly distended and paralytic. A more or less constant dribbling or overflowing of urine may be noted. I have observed a few cases in which obstruction of the urethra was complete. In these cases the animals die rather rapidly unless they are catheterized. A similar weakness of the hind legs also has been mentioned by Hobday.[14]

Treatment of disease of the prostate gland in the dog is dietary, medical, physical and surgical. The prostate gland may easily be palpated through the abdominal wall and rectum. Therefore, a determination of the size and character of the prostate gland is always possible. If the bladder is full, it should be catheterized. Constipation, when present, should be relieved by an enema. The dog should be given a laxative and should receive a reduced diet of meat and cereal. If the dog is obese, fasting for a few days is advisable but he should have access to clean drinking water at all times. Daily massage of the prostate gland appears to be beneficial in some cases. If the urinary obstruction is of long standing, it is advisable to determine the values for the blood urea and creatinine. If the value for the urea is high and if that for the creatinine is not more than 0.3 mg per 100 cc of blood, a good result may be expected. However, when the value for the creatinine also is elevated, a bad prognosis must be considered. Large encapsulated abscesses should be drained surgically when it is possible. Castration has been shown to arrest development of the prostate gland. It also is followed by a reduction in the size of the gland when moderate hyperplasia and hypertrophy are present. This operation may be advisable in selected cases to prevent recurrence of marked enlargement.

Müller[17] advised small quantities of nonirritating food, cold or lukewarm applications to the perineum, frequent catheterizations, warm or cold enemas, saline purgatives, and suppositories of cocoa butter and belladonna. He said that abscesses may be incised through the rectum or ruptured into the urethra by application of pressure. Hobday,[14] French[15] and Brumley[21] also recommended reduced diets, laxatives, catheterization, enemas,

drainage of abscesses, massage, injection of potassium iodide into the prostate gland, and castration in suitable cases.

Total and subtotal suprapubic resection of the prostate gland, as was practiced on man prior to the advent of successful transurethral resection, has not been successful in the dog. Transurethral resection of the prostate gland is the most important advance in surgery of the prostate gland in man to date, but it cannot be practiced in the dog because of the os penis which partially surrounds and prevents dilatation of the urethra. In the dog, the prostate gland does not enlarge as it does in man. In man, most obstruction is caused by the middle lobe of the gland; in the dog, this is poorly developed or absent and does not cause obstruction.

Sparks[22] recently reported his method of partial resection of the prostate gland in the dog. He shaves or slices the tissue from the outer portion of the gland to reduce its size. He does this when marked enlargement of the prostate gland complicates the reduction of the perineal hernia.

At present, many urologists are interested in the newer theories on hypertrophy of the prostate gland, as recently reported by Lower and Johnston,[19] Lower,[20] Moskowicz[23] and others. Much is gained from such investigations. They may revolutionize present methods of treating this disease in man and one may find a treatment applicable to the dog.

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Discussion

Dr. J. V. Lacroix: In Doctor Schlotthauer's excellent presentation of the subject we have been given to understand that the prostate is intimately associated with sexual functions and that this gland is readily influenced by endocrine secretions. These facts probably apply to man and beast alike.

Too little is known regarding the prostate in health and disease in dogs. Authors who have written on the subject have probably drawn from medical literature and in some instances they have presumed. It would be interesting to have dependable information on the behavior of the prostate in dogs of different ages and under various conditions during illness and health. At present we recognize inflammations of the prostate when these occasion serious disturbance. Practitioners generally do not make routine examinations of the prostate. Practitioners generally have little knowledge regarding the applications of the very excellent means of treating some prostatic ailments—massage. In fact, it is said that comparatively few urologists possess this faculty.

Records at the North Shore Animal Hospital (Evanston, Ill.) reveal the fact that diseases of the prostate in the dog occur rather infrequently or that such diseases are infrequently recognized. Furthermore, these records, where affections of the prostate are concerned, indicate that clinicians who make these entries are inadequately informed on the subject. However, these records do indicate that inflammations of the prostate interfere with the elimination of feces and in some instances with the excretion of urine. They indicate that in some animals where the prostate is inflamed and enlarged there is fever. Some animals are toxic and move about with difficulty. In some cases the back is arched and where acute infections exist patients have succumbed within four or five days after the nature of the ailment was first recognized.

We have encountered cases where large prostatic abscesses seem to have caused the death of dogs but these abscesses were found to be encapsulated on autopsy. There was no drainage of pus from the urethra. Unfortunately, we have no record of attempts made to collect secretions, for microscopic study, by "milking" the prostate after massage. However, we do have information indicating that inflammations of the prostate occur without occasioning serious general disturbance and that they yield following the administration of enemas, laxatives and massage.

The following notes are taken from the records on a Cocker spaniel which first came to our attention when it was eleven weeks of age, on December 12, 1931. This animal has been in and out of the hospital many times between the first visit and the period of hospitalization just concluded a few days ago. On February 4, 1932, at 4½ months of age, this puppy was treated for ascarids, receiving santonin, castor oil and enemas. A little later in the month, distemper prophylaxis was

begun. When this puppy was about seven months of age, the clinical record shows an entry, "Obstipation," but there is no record on the condition of the prostate at that time.

On July 21, 1932, the records show that the patient was again treated for obstipation.

On March 5, 1933, this patient was brought to us in a state approaching collapse. There was an intoxication apparently of digestive origin but no constipation existed at that time.

From September, 1933, to April, 1935, this patient suffered, at times, from indigestion, enteritis and dermatitis.

On April 25, 1935, the anal pouches were evacuated but again no entry is made regarding the condition of the prostate.

On August 8, 1935, following several digestive disturbances, three infected teeth were removed. There was no record of ill health thereafter until February 26, 1936, when records show that the dog suffered from constipation and again, for some reason, the anal pouches were examined and the notation made that these were evacuated.

Finally, on July 21, 1936 (two years after the first attack of obstipation), the records show the diagnostic entry, "Constipation and Prostatitis." During the period between July 21, 1936, and August 5, 1936, this patient received enemas, vaseline *per os* and the prostate was massaged several times. Inflammation and enlargement subsided and my entry on August 5, 1936, shows that the prostate is still slightly enlarged, somewhat flabby but not inflamed.

This report is given in rather tedious detail to support the opinion that the prostate should always be examined whenever there is occasion to evacuate the anal pouches and that wherever obstipation occurs in young dogs one should not fail to examine the prostate.

Here is a field for clinical research which ought to yield practical results.

Woodchucks

The fact that Groundhog Day occurs in February, a story about this animal in *Our Dumb Animals* should be interesting.

A woodchuck is not popular with farmers because of its habit of burrowing in fields, destroying crops and endangering live stock which may fall into the holes.

According to the author of the story, the woodchuck has traits which should give it consideration. It will eat corn while it is green but will not touch it after it is ripe.

The main food of this animal is roots and vegetables. It will not eat meat, but will drive rats and mice from its stamping grounds. It has been observed that these rodents are never found in corn shocks which stand near the dens of woodchucks.

Michigan Dairy and Milk Inspectors to Meet

The Michigan Association of Dairy and Milk Inspectors will hold its annual meeting in connection with the meeting of the Michigan Allied Dairy Association, March 10-11-12, 1937, in Grand Rapids, Mich.

THE RELATIVE VALUE OF FARM GRAINS IN POULTRY NUTRITION*

By H. L. WILCKE, *Ames, Iowa*
Iowa Agricultural Experiment Station

Seventy to 85 per cent of the more practical poultry rations consist of grains and grain by-products. For this reason, it is highly desirable that we obtain information as to the relative value of the more common farm grains in mash mixtures. Then, too, the veterinarian is often called upon to diagnose the cause of mortality when chicks or adult birds have been fed rations which are made up of single grains, usually with milk as a protein supplement, but sometimes with no protein supplement whatever. In such cases, there must be some basis for the proper evaluation of the diet. In the past, corn and milk have been considered a quite satisfactory diet, while oats and milk have been considered inferior, or even unsatisfactory because of their relatively high fiber content. The information reported in this paper will aid in forming an opinion of the relative worth of some of the grains when they are used to make up the entire cereal grain portion of the chick ration.

The time allotted to this paper will not permit an extensive review of the literature. The background for this type of experimentation may be found in a previous publication,[1] in which some of the references have been cited.

In this experiment, seven lots were used, six of which consisted of 42 Single Comb White Leghorn chicks each. These chicks were taken from the incubator and banded on the 22nd day. Each lot was then divided into two groups in order to run duplicate pens on each ration. The chicks were placed in battery brooders, and they were fed and watered immediately. A seventh lot, consisting of only 28 chicks, was also included in the experiment, but this lot was not run in duplicate because of the smaller number of chicks available.

The rations which were used are presented in table I, and the calculated analysis of each ration may be found in table II.

The chicks were weighed at bi-weekly intervals until they were ten weeks old.

The weights of the males are presented in fig. 1 and those of the females in fig. 2. It will be observed that there is practically

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TABLE I—*Composition of rations (in pounds).*

INGREDIENT	RATION						
	155	156	157	158	159	160	161
Ground yellow corn	72	81.5	—	—	—	—	—
Ground wheat	—	—	72	—	—	—	—
Ground whole oats	—	—	—	72	81.5	90	92
Dried buttermilk	25	15	25	25	15	5	5
Ground oyster shell	1	1.5	1	1	1.5	2	1
Steamed bone meal	1	1	1	1	1	2	1
Salt	1	1	1	1	1	1	1
Cod-liver oil	1	1	1	1	1	1	1
Totals	101	101	101	101	101	101	101

TABLE II—*Analysis of rations (percentage composition).**

	RATION						
	155	156	157	158	159	160	161
Protein	15.17	13.08	18.94	18.44	16.78	13.88	14.16
Ash	5.30	3.92	5.68	6.98	5.82	4.78	4.54
Fat	3.80	3.78	2.52	4.18	4.22	4.24	4.25
Fiber	1.69	1.95	1.64	7.73	8.80	9.688	9.89
Ca	0.933	0.619	0.964	0.992	0.687	0.414	.350
P	0.566	0.545	0.668	0.612	0.518	.433	.413

*Based upon analyses of ingredients made by the Animal Chemistry and Nutrition Subsection.

no difference between the growth rates of the chicks fed 72 per cent of ground oats and 25 per cent of dried milk as compared with those which were fed 82 per cent of ground oats and 15 per cent of dried milk. The mean weight of the males in lot 158 was 689.7 gm, as compared with a mean weight of 664.6 gm for the males in lot 159, but in the case of the females, the direction of the difference was reversed, that is, the females in lot 158 weighed 613.3 gm, as compared with a mean weight of 625.0 gm for the females in lot 159. This difference was not significant in either case, indicating that 15 per cent of dried milk is just as effective as 25 per cent in supplementing ground whole oats, but there is a possibility that some intermediate level might produce better growth. These growth rates are quite comparable to those which have been published previously.[1]

The chicks which were fed 90 per cent of oats and five per cent of dried milk, with two per cent of oyster shell and two per cent of bone meal, grew somewhat more slowly, as was to be expected,

but the weight at ten weeks was greater than that of the chicks which were fed 82 per cent corn and 15 per cent milk. These results indicate that oats is much more satisfactory for growth than either corn or wheat when these grains are used in single-grain rations.

Probably the most striking differences obtained were those between lots 160 and 161, both of which received the same amount of dried milk, the difference being in the amount of ground oyster shell and steamed bone meal in the ration. Lot 160 received 2 per cent of each of these products whereas lot 161 received only 1 per cent of each. At ten weeks of age, the males in lot 160 had

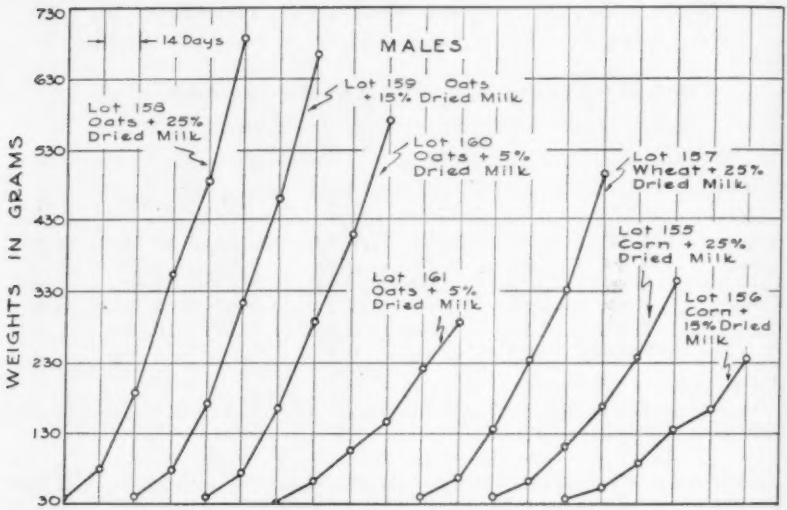

FIG. 1. Graph showing weights of male birds.

a mean weight of 568.95 gm, as compared with a mean weight of 286.43 gm for the males in lot 161, and the mean weight for the females in lot 160 was 516.32, as compared with 285.42 gm for the females in lot 161. There was also a marked difference in the appearance of the chicks of the two lots. The chicks in lot 160 were smooth, well-feathered, and thrifty in appearance, while those in lot 161 appeared to be rachitic. In order to determine whether these chicks were actually rachitic, bone ash determinations were made on the tibiae of five representative males and five representative females. The mean bone ash was found to be 35.73 per cent with a standard deviation of 2.36 for the males, and 34.64 per cent with a standard deviation of 1.52 for the females. These are definitely rachitic values. Obviously, the calcium content of ration 161 was definitely lower than the percentage which is re-

quired for normal growth and calcification in Single Comb White Leghorn chicks. Ration 160 was also low in calcium, but no clinical rachitis developed in this lot. No bone ash determinations were made for lot 160.

The growth on the two corn rations was poor, as may be observed in figs. 1 and 2. There were also many cases of perosis in these groups. Eighty-seven per cent of the chicks fed 72 per cent of corn developed cases of perosis, and 97.6 per cent of those fed 82 per cent of corn were affected with this condition. In the majority of cases, the condition was so severe that these two lots

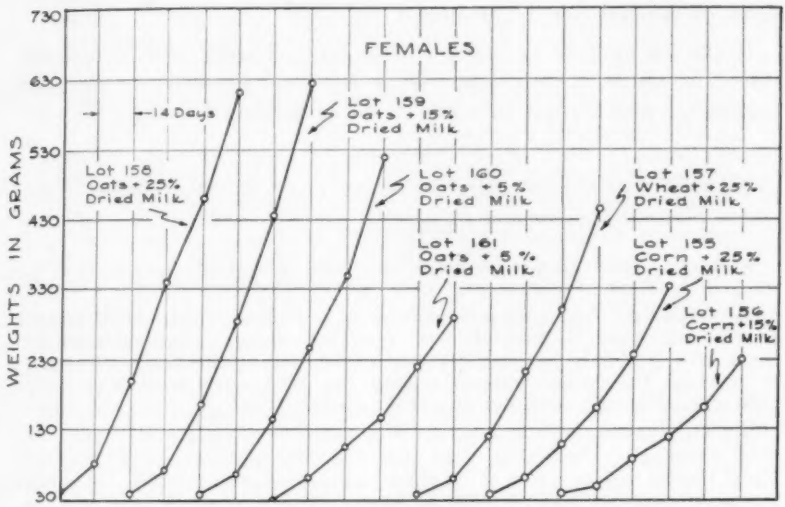

FIG. 2. Graph showing weights of female birds.

were discontinued when the birds were ten weeks of age. The feathering of these birds was slow and of poor quality, in sharp contrast to that of the chicks which had been fed oats or wheat.

It is obvious that the differences in the protein content of the rations were not responsible for the differences in the growth of the chicks on the various rations. This is brought out by the fact that the growth of the chicks in lot 157, which received the ration highest in protein, was less than that for lots 158, 159 and 160, which received rations lower in protein, and also by the fact that the chicks in lot 160 made better growth than those in lot 155, even though ration 160 was lower in protein. Therefore, it appears that there is some constituent in oats, and to a lesser extent in wheat, which is necessary for good growth and good feathering in chicks, but this substance is lacking in corn.

SUMMARY

Rations consisting of large amounts of ground whole oats as the sole source of cereal grain have been found to be quite satisfactory for growth and feathering in chicks. A ration containing 81.5 per cent of ground whole oats, 15 per cent of dried buttermilk, 1.5 per cent of ground oyster shell, 1 per cent of steamed bone meal, 1 per cent of salt, and 1 per cent of cod-liver oil produced good growth and excellent feathering in Single Comb White Leghorn chicks. A ration containing the same amount of corn with the same amounts of these supplements, was not satisfactory from the standpoints of growth, feathering or the number of cases of perosis which developed.

There appears to be some constituent in oats, and to a lesser extent in wheat, which is necessary for good growth and for good feathering chicks, but this substance is lacking in corn.

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DISCUSSION

MEMBER: How do you prepare your oats for this mash to feed the chicks?

MR. WILCKE: The oats are ground in a hammer mill; they are not ground particularly fine. If you feed the mash in an open hopper, there will probably be considerable waste; but if the hopper has a lip to prevent the birds from dragging the feed out, there will be no difficulty, and they will eat it quite readily.

DR. H. E. ASH: How long do you keep them on the ration?

MR. WILCKE: We have kept some of the birds through one complete laying season, and it worked out very satisfactorily. We have not been doing it long enough to operate it any further than that yet. I might mention that we start them on the ration when they are a day old, and they are taken direct from the incubator and put in these pens.

DR. ASH: How do you find them with regard to disease? Do you have more or less trouble than you would on the other rations?

MR. WILCKE: The mortality has been very high on all rations; but it has not been higher on the oats than on the others. However, it has been too high on all of them. The first-year mortality has been a distinct problem with us, as it has been with other poultry people, and a mortality of 35 to 40 per cent is not uncommon in laying pullets. That is why I say it is too high in all the rations. However, we do encounter more difficulty with the mortality factor in the wheat group than we do in the others.

DR. W. A. HIGGINS: Did you raise these and keep them through the laying season in hen cages, or did you put them in pens?

MR. WILCKE: They were put in pens when they were twelve weeks old. They were on wire when they were outside.

DR. B. A. BEACH: Have you encountered or had any experience with, or made any observations on gizzard lesions—erosions of the lining of the gizzard—with respect to these various rations?

MR. WILCKE: No. However, all of these birds have been autopsied and no reports have as yet been returned from the diagnostic laboratory. I do not know whether they examined them for that particular thing or not.

DR. BEACH: We have encountered that in Wisconsin to quite an extent. We do not know what the significance of it is. It is a pathological condition; it is certainly not always fatal; and how important it is we do not know. However, the addition of "midds" and bran will prevent that, and I do not know whether the experimental work has gone far enough to enable us to be sure about that. I was just wondering whether anyone else has made any observations on it.

MR. WILCKE: I think Dr. Bethke has done something on that. Dr. Bethke, would you give us some information on the gizzard lesions in chickens? I think you were talking about that some last week, were you not?

DR. R. M. BETHKE: Yes. We have encountered the condition any number of times on any variety of different types or kinds of rations. We do not know specifically what will prevent the condition. There are some reports of work done in Wisconsin which indicate, or rather suggest, that it is a vitamin-like factor.

We might have that situation, for instance, in the breeding. Apparently, from present indications, you will have a considerable amount of this material in the green grass, the succulent material, and more so than in the grasses which are dried again. There seem to be certain other classes of compounds, like mucus tissue, and things of that type, which seem to be especially good sources of this factor, according to the Wisconsin authorities.

DR. A. T. THOMPSON: Do you feed any grit with these rations?

MR. WILCKE: No, we did not. There was no grit; it would be an all-mash ration. The only things we had in the ration were those I gave you, plus water; and that was their feed.

I might say that we have had no trouble with any nutritional developments, except, as I indicated in that one group of chicks which had been fed on wheat rations. We are continuing to feed those birds in that manner for a year's egg production so we can get a better check on that.

DR. E. D. HUBBARD: How much mortality do you have to twelve weeks?

MR. WILCKE: In some cases, none; in one lot, we lost five chicks of the 21. That was in one of the corn groups; but in the duplicate lots we lost none whatever. That is the advantage of operating duplicate lots. There was no mortality difference which we could attribute to any ration.

Care of Cats

Dr. Hugh F. Dailey, Chief Veterinarian, Angell Memorial Hospital, Boston, Mass., has given some good advice for the care of cats during cold weather in an article appearing in the February issue of *Our Dumb Animals*. The writer points out that cats inured to an indoor life have lost their resistance to cold, so cannot withstand sudden weather changes to which alley cats have adapted themselves. To the house cat, there is a danger of frozen pads or pneumonia if compelled to remain out in the cold weather for a considerable time.

FIELD OBSERVATIONS ON THE INCIDENCE OF FOWL LEUKOSIS IN HATCHING EGGS AND CHICKS*

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University of Wisconsin

The term fowl leukosis, for purposes of this discussion, will be used to designate both the lymphomatous and fowl paralysis types of the disease, or diseases, as the case may be.

Some doubt concerning the transmissibility of fowl leukosis through hatching eggs has been raised recently. Mention of some of the literature bearing on egg and chick transmission may not be out of place, though a complete review will not be attempted at this time. Biely and Palmer[1] and Johnson[2] have given rather complete bibliographies on fowl paralysis and lymphomatosis, respectively.

According to Warrack and Dalling[3]:

> Various observers have voiced the opinion that fowl paralysis is transmitted from the adult to the chick *via* the egg, and that the incubation period is two months or more. Doyle (1927) says: "We have reproduced the disease by merely breeding from an experimental flock in which there had been cases of paralysis, and in which there were cases of iritis. Several of the birds hatched from eggs laid by the affected flock developed typical paralysis with well-marked neuritis and myelitis before they were two months of age—one at 47 days. Control birds from a flock in which the disease was never known to have occurred have not shown evidence of neuritis or paralysis during a period of six months. * * * We have repeatedly observed the disease to occur for the first time in flocks following the introduction of birds from affected flocks. We have also observed the disease to develop in birds which were obtained as day-old chicks from affected flocks, and then raised in new houses on new ground. The observations which we have made regarding the spread of neuritis or paralysis in the field indicates that the important factor in its dissemination is the sale of breeding stock, baby chicks or eggs for hatching, from a flock in which one of several birds have shown symptoms and lesions of this disease."
>
> Marginson and McGaughey (1931) refer to fifteen outbreaks, in nine of which there was strong evidence that infection was introduced through eggs, and in six by young chicks. Commenting on their observations, they say: "We do not intend to imply that this method of spread is the only possible one, but desire to point out its importance. It would appear that the principal method of spread is through breeding from affected adult birds."
>
> Bayon (1931), referring to preventive measures, says: "Last, but not least, the source of the strain affected must be considered and scrutinised, and if necessary avoided in the future." Beaudette

*Presented at the seventy-third annual meeting of the American Veterinary Medical Association, Columbus, Ohio, August 11-14, 1936.

and Hudson (1931) state that the appearance of the disease on a new farm is always preceded by the introduction of hatching eggs, baby chicks, or breeding stock, and that it is always possible to trace new outbreaks to introduction of eggs, etc., from an infected farm. They conclude by stating that "while there is much to be done before the question of transmission can be finally settled, the evidence is strongly in favour of the conception of transmission through the egg."

Again quoting Warrack and Dalling[3]:

We have had the opportunity of carrying out a laboratory experiment in connection with egg transmission. From a heavily infected flock three consignments of eggs were obtained and hatched by us in our own incubators. The chicks were subjected to various conditions; in six birds typical fowl paralysis developed. This carefully controlled experiment adds more evidence to support the contention that fowl paralysis may be egg-transmitted, and that the association of intestinal parasites is not essential.

Example of One Bird in a Flock Producing Affected Progeny

In a flock of Rhode Island Reds was hen 115, which had an excellent egg record. She was bred, and 74 of her eggs were hatched and yielded 59 chicks. Between two and five weeks after hatching, several died from an undiagnosed cause. Fourteen of the remainder were rung and therefore easily recognized; of them, six died at ages between five and seven months. No other birds have died on the farm during the present season. We had opportunity of examining four of the six birds. In two there were swollen nerves with the usual microscopic picture. One had a typical ovarian tumour, and in the other was a huge tumour under the skin of the neck. The cells composing these tumours were typical of fowl paralysis. It is of interest to note that in no case was there any evidence of disease of the duodenum, and the only parasites found were a few coccidia in the caeca on one bird. It seems evident that the disease was transmitted by hen 115, and not by the male birds, as none of the progeny of any other hen has shown any signs of disease.

A private communication from Dr. R. Fenstermacher, under date of February 8, 1936, describes a hen in his experiment flock, identified as D 71.

This bird was first placed in the experiment during 1933. That year she produced 19 chicks that were reared beyond six months of age. The following figures you will observe refer only to birds reared beyond six months of age. The six-month period was used as an arbitrary age. Of the 19 birds that were reared from the hen that year, none of them developed lymphocytoma while seven developed fowl paralysis. Her history was as follows: Three of her brothers and sisters out of a group of five died as a result of lymphocytoma.

The following year, 1934, we reared twelve chicks from this hen. Four of this group died as a result of lymphocytoma while two developed fowl paralysis. During the year 1935, she produced nine chicks, none of which developed fowl paralysis and none lymphocytoma. Of course some of these birds are still in the experiment. They may develop either disease later on.

Both diseases continued to appear in the F 1 and F 2 birds reared from this hen. It is true that the F 2 group which is in our possession at the present time does not show as much evidence of the disease as it was in the F 1 group. However, I do wish to state that if one kept birds in a breeding flock which originated from families where either of these diseases was present, it is my opinion that one had better have an income from other sources, as the losses will be rather severe. The losses from intermittent diseases have also been heavier in this group than in our control group reared under similar conditions. At the time that D 71 was autopsied, we found no lesions indicative of either disease under discussion.

Our Own Observations

Farm 1: The owner purchased 100 Barred Rock chicks and 400 White Leghorns. These chicks were raised in the same brooder under identical conditions. As a matter of fact, they were all running together. He raised 99 of the Barred Rocks and only 60 of the Leghorns to maturity. There was a bad outbreak of range paralysis. During the first year, approximately one-half of the 60 went down with the disease, making a total of 30 saved out of 400. None of the Barred Rocks contracted the disease.

Farm 2: Five hundred Single Comb White Leghorn chicks were bought, 100 from each of five different hatcheries. The owner made pens for them in a barn that had been used previously for cattle. This barn was repaired and thoroughly cleaned and disinfected. He kept 100 chicks in each of the five pens. A bad outbreak of range paralysis occurred in one of the lots of chicks, and this lot was from the same hatchery whose chicks suffered most of the outbreaks of the disease in that community last year. As a matter of fact, they were from the same hatchery whence the chicks on farm 1 were procured.

The following observations were made in one Wisconsin county:

Farm 3: Fowl leukosis had never been seen or heard of on this farm until the poultryman purchased 225 Single Comb White Leghorn chicks from a certain hatchery. These were placed on clean ground in a new brooder-house. At about eight weeks, some of the chicks became paralyzed, blind, or both. In October, when we visited the farm, 60 of the 225 birds were left, and fully half of them were manifesting clinical symptoms of leukosis.
graph 1 of Section 588, Postal Laws and Regulations, specifically

Farm 4: Here both Single Comb White Leghorns and Barred Rocks had been purchased from two different hatcheries. Prior to the spring of 1935, the hens on this farm had never exhibited symptoms of fowl leukosis. The disease appeared in both breeds of chicks, and the owner's losses that spring and the following winter were fully one-half of the birds. Whether both lots were

infected from the hatchery, or one lot became infected from the other, we do not know.

Farm 5: Fowl leukosis appeared in the chick flock on this farm during the summer of 1935. This was the first appearance of the disease here. These chicks came from several different hatcheries.

Farm 6: About half of a small hen flock on this farm was lost during the winter of 1935. The chicks were farm-hatched from the owner's eggs. The only birds introduced into this flock were some brooding hens from a flock known to be infected with leukosis.

Farm 7: One hundred and twenty-five chicks were purchased for this farm in the spring of 1935. About half the flock succumbed. Many of the cases were definitely diagnosed as leukosis. This was the first appearance of the disease on this farm.

Farm 8: This farm was operated by the son of the party owning farm 7. He had the same experience with chicks purchased from the same hatchery, and experienced a somewhat heavier loss.

Farm 9: One hundred hatchery chicks were bought for this farm. At about eight weeks of age, they started to exhibit symptoms of leukosis. About 40 died during that summer and the following winter.

Farms 10 to 13: In an adjoining county the following observations were made: Five farms were visited. On four of these, which we shall designate farms 10, 11, 12 and 13, the owners had purchased chicks in which fowl leukosis developed with losses varying from 10 to 50 per cent. These chicks were bought in the spring of 1935, and in all four cases represented the first appearance of the disease as far as we could determine on these farms.

Farm 14: An interesting observation was made here. Chicks were procured during the spring of 1935 from the same hatchery supplying many of the other chicks. They were brooded in the usual manner with a small loss. The hens went through the winter of 1935-36 with an unusually low death-rate. As far as the owner knew, these hens were free from disease. Chicks were hatched from this flock in the spring of 1936. At about eight weeks of age, some of them commenced to show symptoms of fowl leukosis, and up to August 4, 1936, about 20 of them had succumbed. On the above-mentioned date, the hen flock was carefully examined and fowl leukosis definitely diagnosed. Evidently these hens carried the virus, but the progress of the disease was slow. They did, however, transmit the disease to their chicks, which exhibited a much more virulent form. The chicks

were brooded in a meadow entirely away from the hens. That the disease might have been transmitted directly from the hens after the chicks were hatched is admitted.

It should be understood that the mortalities given may be far from accurate. It was necessary to take the owner's word, and other causes might have entered and probably did enter in without his knowledge. Only those farms on which a positive diagnosis of fowl leukosis by a competent person was made are included in this survey.

Summary and Conclusions

Fowl leukosis was studied on 14 farms where the disease had not appeared until the spring of 1935. In all cases symptoms first appeared in chicks. In twelve instances the chicks came from three hatcheries; in the two others the chicks were hatched from eggs produced on the farm. One of the latter hen flocks was shown to be infected, but in the other flock the source of the infection was not determined.

Seven of the observations were made in a county where a field veterinarian on the State Veterinarian's staff had recently made a farm-to-farm canvass of the poultry-disease situation. Acknowledgment is hereby made of the valuable assistance given.

References

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[2]Johnson, E. P.: A study of lymphomatosis of fowls. Va. Poly. Inst. Tech. Bul. 44.

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Discussion

Dr. Hugh Hurst: I would like to ask if anyone has any data which would indicate that birds hatched from hen eggs, of birds three years old or older, are less susceptible than chicks hatched from pullet eggs?

Dr. F. D. Patterson: I am sorry to say we have no information in regard to whether or not pullet eggs are more liable to transmit the infection. One thing I might mention is that if the older bird has not shown any signs of leukosis by the time it is three years old, the chances are it is not infected. We have kept them over quite a few years, but most of those birds become non-productive.

With the original experiments, it looked as though it was transmitted only through the eggs, because you could trace it right back through the flocks in the hatcheries. However, when you put it on a basis of controlled experimentation, it seemed that the most common mode was by direct contact with infected birds, and by running on contaminated ground. The disease was apparently contracted by most of these birds when they were young, or at the time they were chicks.

It seems to me the major point in the matter of transmission through the egg, according to our experiments, is the inherited transmission of susceptibility or resistance. You can find certain strains of chickens, or certain individuals, whose chicks are much more susceptible, or much more resistant, than others.

To my mind, one of the most important problems which is yet unsolved, as far as leukosis is concerned, is the transmission of the virus. We think—we do not know—that under certain conditions the virus of leukosis is transmitted through the egg; but as far as the individual instances in the birds are concerned, apparently most of them contract it after they are hatched, at an early age. However, you will find birds with all stages of the infection in the ovary and some in the intestines, where we have lymphomatosis. In those few instances where production is not completely checked at an early date, where the generative organs are involved, it seems reasonable to expect, in a few cases, it might be transmitted through the egg.

Here is another point. If only one chick out of a thousand or more should happen to contract the disease through the egg and was exposed to a large flock of very highly susceptible chicks, that would certainly constitute a major problem. If it is transmitted at all through the egg it is not frequently of major consequence. Our experiments, when you control them, seem to indicate that is not so important a mode of transmission as we thought. When we first began, we thought it was the only method.

DR. BEACH: It would seem that the previous history of the area might have something to do with that. In sections of Wisconsin, where the disease was unknown before 1935, in most instances, it came from hatchery chicks. Whether they contracted it in the hatchery before they left it, or whether it came through the egg, we do not know, but the first appearance on the farm was in the hatchery chicks. Of course that, from the standpoint of control and from the standpoint of the practical poultry man, is the important thing.

DR. R. FENSTERMACHER: We have been studying this problem, which is certainly a serious one, rather closely for four years.

I had a bird, one of a flock of 700, which rats destroyed, and a complete histo-pathological examination was made of all tissues and organs, and there was no evidence in the organs. That was an instance of a bird which had been producing susceptible specimens right along, and then all of a sudden producing non-susceptibles; and that will hold true. I have birds in that flock which are four years old, which have never produced a susceptible bird, even when crossed with a susceptible, or a potentially susceptible male bird, one which comes from a family of susceptibles. Likewise, you can take a group of sisters of a bird and part of the sisters will produce young which are non-susceptible and part of them will do the opposite. The same will hold true in that you will have a bird which will produce non-susceptible birds for a year; the next year she will not produce them and her offspring may be susceptible or they may not.

As Dr. Patterson said, one of the first things that happens is they become non-productive, and when that happens they are, of course, elements which are eliminated from your experiment. They cannot help you. Our results are not comparable to those reported by many workers with this disease.

Just briefly, I will say this: That when these chicks are about four weeks of age—I am speaking now of the controlled ones which come from a flock over which I have close supervision, a large flock of approximately 3,000 producing birds—the incidence of either of these conditions is practically nil. I think there has been one or two cases in the last three years. It is very, very low and probably consists of spontaneous cases. We feel quite safe with our control birds. They are purchased as day-old chicks from the hatchery. When they are about three or four weeks old, they are put into physical contact, right in the same pens, with our potentially susceptible birds, those which we expect to develop either one of these conditions. As far as the practitioner is concerned, it does not make any difference whether

it is fowl paralysis, leukemia, or what it is, because they die; and we have had practically no spread through these contacts. Out of some 1,500 birds reared—each year we rear approximately 500 and I place about 100 controls in the setup—in the time we have had them, there have been less than half a dozen birds of our control groups which developed the disease. I cannot explain it. Whether we have a strain in our district which is not susceptible to transmission in that manner, I do not know.

Another interesting feature of this matter is that originally, when this experiment was begun, there were specimens obtained from a flock which had been pedigreed for years. The birds which we selected came from families—I am speaking now of the susceptible group—in which one or more of the brothers or sisters of those birds developed the disease. In this particular case, the disease was confined entirely to the enlargement of the internal organs—liver, spleen, kidney and ovary. There was no lameness, no paralysis, and no blindness. The very next year, when those birds came to our laboratory—and it is less than 100 feet away, because they were selected from the University flocks—fowl paralysis appeared. Even today, after four years, fowl paralysis is not a problem in the University flock; in my own experiment group it is about 50-50. Where we have birds which become lame or blind, the autopsy will show the internal organs—liver, spleen, kidney, ovary—showing no gross lesions; and many of them have been examined histopathologically and the results have been negative. When they were brought over, those organs remained normal, but we had lame birds, blind birds, and birds with their wings drooping. Why that happened I do not know. I have no explanation and I do not want to offer any, nor do I want to leave any impression that I know what has happened, because I do not. At the present time, this work is continuing, and we are in hopes of producing resistant birds. That is what we are after. However, as I said before, the moment you get a hen infected with this type of ailment, which commonly infects the ovary, she quits laying and she is out of your experiment, and you are finished.

DR. PATTERSON: I would like to say just a word in connection with Dr. Fenstermacher's results, which are quite interesting. It is altogether possible that we might be dealing with different strains in chickens. It appears that there is a type of leukosis which seems to vary greatly with different strains and individuals.

For instance, we had a strain of Rhode Island Reds. I do not want you to misunderstand me, because I do not say that a Rhode Island Red is more resistant than any other breed of chicken. We happened to secure one strain of Rhode Island Reds, as compared to the strain of Leghorns which we are using, and it was very hard to transmit leukosis, except by an injection of a virulent virus. Approximately 50 per cent of the injected birds came down with it, and a much smaller number of the pen-contact birds. We used a less virulent virus, as indicated by the fact that it was more chronic in nature and also by the reaction on the birds. Our highly susceptible Leghorns would come down with it to the extent of 60 to 70 per cent, but no pen-contact birds at all among the Rhode Island Reds. Our experiments were almost positive proof that it was transmitted by direct pen contact and that constitutes one of the most important factors.

We also conducted experiments on transmission through the egg in which every bird in the pen had positive evidence of fowl leukosis. Most of those were confined to eye and nerve types. We found that by keeping those having the eye type for two years that practically every one of them that did not die from some other complication would

die with a visible organ type of leukosis, or lymphomatosis, or something of that kind.

DR. E. P. JOHNSON: I just want to say that I think we ought to be clear in our understanding as to whether, when we talk about transmission through the egg, we mean the virus is being transmitted, or the susceptibility is being transmitted. I am not quite clear, from Dr. Beach's talk, as to whether he intimated that the virus is transmitted or whatever he considers the agent which causes the disease —through the egg, or whether he considers there is a tendency or susceptibility transmitted there.

If the virus is transmitted, it seems to me that birds which are sick and have lesions develop in the ovary would be classified differently in some respects. As you all know, they practically all stop laying at that time. That seems almost to exclude the rapid increase from those birds, as far as the virus is concerned, but as far as the tendency or susceptibility being transmitted, that, of course, could occur in normal birds—the susceptibility for contracting the infection.

There seems to be more or less of an indication that this agent is, I might say, universally present. That is my conception of it; and that there is a tendency which is transmitted. Maybe that is transmitted genetically—I do not know—but certainly certain strains of birds are more susceptible to it than others. However, if we think of it in that sense, we should not really, to be scientifically correct, speak of the agent being transmitted through the egg. The result is the same, but it is just a little different concept of it. That might explain why, if you take these birds to a farm—that is, birds from a free flock to a contaminated flock—you get the incidence of the disease, because the virus is there. On the other hand, if you take resistant birds to that farm where the infection is, you may not have any trouble. That also explains the situation where you have some of these injected birds and put them in a cage along with clean birds. If you have resistant birds, you have no trouble, but if you have susceptible chicks, it is a different story.

DR. BEACH: I evidently did not make my position clear; but what the article reports is this: The incidence of this disease in day-old chicks on farms where it never appeared before in so far as we could determine. Those are facts and the names and the addresses of the men are available. You spoke about the virus transmission through the egg. I do not know about that. It either comes through the egg, or the day-old chick gets it very soon after being hatched. In my mind there is not any question about that. The article cites a number of instances of control experimentation which show that. We have either got to say that these men did not know what they were doing or else we have to accept it.

DR. JOHNSON: Possibly those chicks could have picked it up after they got there?

DR. BEACH: They could not on these farms where it never occurred.

DR. JOHNSON: I am not so sure about that. The virus may be on the place even though there has not been any incidence of the disease.

DR. BEACH: That might be; I do not know; but what about this instance where the only outbreak we could find was in the pullets which were shipped there, and it was confined to that place? In twelve of the 14 farms mentioned here, it occurred in the chicks. There were two that I excluded. It occurred in the chicks and was not present in the old birds, and never had been as far as we could determine.

The transmission of an inherited susceptibility is, of course, admitted. That undoubtedly occurs in all disease as far as we know. The transmission of the disease through the egg, if the facts shown in other diseases hold true, namely of carriers, is possible. But again, I do not know. I will be surprised if, when the last word is said on

fowl leukosis, the virus is not transmitted through the egg. It may be transmitted by birds which have never shown symptoms of the disease, and possibly birds which have not shown lesions either, because we know the carrier problem, especially with the respiratory diseases, is such that the disease may be carried by birds which have completely recovered as far as clinical evidence is concerned. We simply reported on what we observed. If our observations are not correct, you will have to accept them or reject them just as you wish.

DR. M. W. EMMEL: Were these chicks from pullorum-free flocks?

DR. BEACH: In the majority of cases they were not from pullorum-free flocks. Some of the flocks had been tested and the reactors taken out; but the next test, in a number of instances, revealed a certain percentage of reactors. Our results in Wisconsin are similar to Dr. Burriss' in this state. We have been unable, in a limited amount of work, to free flocks with any degree of regularity by the test. We do reduce, in a number of cases, the percentage of reactors, but we do not make them free, and no account was taken of that in the observations made.

CHAIRMAN DOWNS: Many of these problems are yet unsolved, but I feel with the high degree of enthusiasm and determination shown in this meeting by the type of men we have represented that in the near future at least many of them will be worked out.

Electric Eye for Grading Milk

The "electric eye," known technically as a lactochromometer, is a device for grading milk according to its color. It was demonstrated at the meeting of the International Association of Milk Dealers at Atlantic City, N. J., on October 12-14, 1936, by Dr. John W. Bartlett, of the New Jersey Agricultural Experiment Station. About 1,500 cows in New Jersey and surrounding states are now being subjected to milk color tests. Dr. Bartlett stated that specimens of milk from related cows, especially in registered herds whose lineage can be traced definitely, are being compared to prove or disprove the theory that desirable milk-color traits are passed from dam to daughter. These tests are being made for the benefit of milk-producers who are desirous of maintaining a milk supply of uniform color throughout the year.

Meat Consumption

The United States has less than one-sixteenth of the world's population, but the country consumes about one-fourth of all meat used for food, Frank A. Hunter, East Saint Louis, Ill., chairman of the board of the Institute of American Meat Packers, stated at the thirty-first annual convention held recently in Chicago. The annual consumption per capita since 1900 has averaged about 62½ pounds of beef, 6½ pounds of veal, 63½ pounds of pork, and 13 pounds of lard.

REGULATING THE SALE AND USE OF BIOLOGICAL PRODUCTS CONTAINING PATHOGENIC ORGANISMS*

By H. M. KALODNER, *Harrisburg, Pa.*
Director, Pennsylvania Bureau of Animal Industry

The Bureau of Animal Industry, Pennsylvania Department of Agriculture, is a regulatory body and as such is charged by law with the prevention, control and eradication of transmissible diseases of animals and poultry.

In Pennsylvania, the biological law, which was approved July 22, 1913, states:

> It shall be unlawful for any person to manufacture for sale or sell or offer for sale any biological product intended for diagnostic or therapeutic purposes with animals excepting upon specific permission so to do from the Pennsylvania Bureau of Animal Industry or unless such product is officially endorsed by the Bureau of Animal Industry of the United States.

The virus act, approved April 27, 1909, states:

> No person shall inject into or otherwise administer to any domestic animal that is producing or that is to be used as food for man any virus or other substance containing pathogenic or disease-producing germs of a kind that is virulent for man or for animals excepting upon specific permission so to do from a member or authorized agent of the Pennsylvania Bureau of Animal Industry.

It is, therefore, apparent that the sale, offering for sale and injection into domestic animals of biological products, without written permission from the Bureau of Animal Industry, is contrary to law.

The question has been raised that inasmuch as the biological law makes the exception for products endorsed by the United States Bureau of Animal Industry, all biological houses operating under federal permit are exempt from the provisions of this act. It is our understanding that the United States Bureau of Animal Industry does not officially endorse any biological product and that a permit to manufacture such products cannot be defined or construed as an endorsement.

Prosecutions have been instituted by the Pennsylvania Bureau of Animal Industry in violation of this act and convictions obtained; the opinion of the judges of the courts being that the burden of proof is upon the defendant that the products sold without permission from the Bureau of Animal Industry were officially endorsed by the United States Bureau of Animal Industry. We contend that if the United States Bureau of Animal In-

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dustry officially endorses any biological product or group of products, that the names of the manufacturers and the name of the biological product so endorsed should receive official recognition and that all states where these products may be sold shall be advised of the official endorsement by the United States Bureau of Animal Industry.

The Pennsylvania Bureau of Animal Industry has been confronted with a serious problem in connection with the illegal distribution and sale of poultry biologics, also the establishment of short-course schools, the purpose of which is to instruct and educate poultrymen, feed dealers and interested laymen in the diagnosis, treatment and postmortem pathology of transmissible diseases of poultry. This Bureau contends that the veterinarian is the only person professionally equipped to help the poultryman to cope with his disease problems. He alone has received fundamental training in transmissible diseases.

The basic subjects covered in the course of veterinary medicine, such as anatomy, physiology, physical diagnosis, surgery and therapeutics, have fitted graduates of schools of veterinary medicine to cope with any disease problem that may arise.

We feel that the land-grant colleges should take care of poultry husbandry, but the diagnosis, prevention, control, eradication and treatment of diseases of poultry as well as other classes of live stock should come within the province of the licensed practitioner of veterinary medicine.

The promiscuous sale and unwarranted use of biological products, the therapeutic value of which is questionable in many cases, have been marked factors in the dissemination of transmissible diseases. This fact becomes more and more apparent from the numerous letters received, especially from poultry-owners who have unwittingly been inveigled into having their flocks treated for some hypothetical disease condition with a biological product of negligible value.

Although products shipped interstate come under the jurisdiction of the federal government, we feel that if the respective states have the right to specify the health requirements of animals entering such states, they have an equal right to specify what biological products may be brought into the states, as well as the disposition and administration of same.

The Pennsylvania Bureau of Animal Industry is of the opinion that vaccines and viruses are disease germs and, therefore, the above-mentioned postal regulation is applicable and the above-

mentioned products are non-mailable matter under the provisions of the act unless mailed to the above-mentioned persons.

We maintain that it is a violation of the postal laws to transmit vaccines and viruses through the mail, inasmuch as paragraph 1 of Section 588, Postal Laws and Regulations, specifically states:

> All disease germs or scabs are hereby declared to be non-mailable matter and shall not be conveyed in the mails or delivered from any post office or station thereof nor by any letter carrier, but the Postmaster General may permit the transmission in the mails under such rules and regulations as he shall prescribe, as to the preparation and packing, etc., providing that the transmission in the mails of the above-mentioned products may be limited by the Postmaster General to shipments of such articles from the manufacturer thereof or dealers therein to licensed physicians, surgeons, dentists, veterinarians.

The Pennsylvania Bureau of Animal Industry would like to recommend to this organization for its approval and action a resolution calling upon the federal government to coöperate more fully with the respective states in order that this nefarious practice of interstate traffic in worthless biological products may be stopped.

Marsh Rabbit

Because of a prejudice against anything containing the name of "rat," the term "marsh rabbit" has been applied to the meat of muskrat, according to the *Black Fox Magazine*. The flesh of the animal for human food is variously esteemed, with diversity of opinion as to its palatability. The meat is usually available from December to March, during the trapping season. The muskrat furnishes a cheap meat, as it may be prepared for food with very little additional labor, after the pelts are removed from the animals.

Certified Cream to Moscow

The new quick-freezing method developed by Clarence Birdseye, while working with the U. S. Biological Survey in 1925, has made it possible for Mrs. Joseph E. Davis, wife of the American Ambassador to the Soviet Union, to have sent to Moscow, 2,000 pints of certified cream. According to *Certified Milk*, Dr. J. G. Hardenbergh, of the Borden-Walker-Gordon Laboratories, is authority for the statement that the cream would remain fresh for two years in the water-proof, cardboard containers in which it is to be shipped.

THE GRADING OF VETERINARY COLLEGES*

By L. A. MERILLAT, *Chicago, Ill.*

One who will trouble to review the decisions of the American Veterinary Medical Association since it was founded, about three-quarters of a century ago, will be agreeably surprised at the absence of mistakes made in the handling of the more perplexing problems strewn along the rough and rugged road it has had to travel through unexplored ground. As stated in "Veterinary Military History," just off the press, it has often hesitated but never erred in the making of final decisions. An example of one of these "more perplexing problems" is the grading of the present colleges of veterinary medicine, which was postponed at the Columbus (1936) meeting by action of the House of Representatives, after quite a heated discussion over the report of the Committee on Education, which, not without good arguments, had recommended that the schools be placed in two classes. The discrimination was based upon teaching facilities and the number of teachers. The length of the curriculum and matriculation requirements, perturbations of the Association up to the time of the World War, are settled questions since the private-school period passed into history and the exclusive state-school period began as the smoke of the World War was clearing away.

While the action of the Association at Columbus may seem reactionary, it was but the echo of the "managed progress" that has characterized the work of the national organization throughout its long career. One must venerate the American Veterinary Medical Association for its conservative ways, for its realization that its cardinal project is to fashion the veterinary service to the best advantage of the American Republic. In keeping this first principle uppermost in mind, the Association has been quite constantly criticized for inaction by its component groups: educational, clinical, public service, etc. The obligation was to build up a profession from nothingness, through the sickly impotence of its early life, to whatever state of healthy strength it now enjoys. The more thoughtful leaders of the organization never lost sight of the fact that the first step in constructing a veterinary profession was to somehow create a personnel and thereafter to maintain it in sufficient numbers to mobilize some degree of affluence. The chaos and quackery, stretching back as far as American history, had to be overcome by first educating a personnel *as well as the offerings justified.* There was no precedent

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to hold out as a bait, no alluring destiny to promise young men, nothing but a forbidden realm degraded by charlatanic adventurers, in short, a low occupation for respectable folks to enter. Much of this has been lived down, but too much still lives.

Although the whole history of the veterinary profession is extremely short, the inclination to look back to analyze what has gone before seems lacking in our councils. The profession we cultivate is but a few years old. Living veterinarians have spanned it all. For the sake of argument, set the birth at 1885. A year or two in either direction does not matter. To say that there was a veterinary profession in the United States before the decade halved by 1885 is stretching the imagination. At that time, a scattered few were straining to form one but without making much headway. The national association drew a "crowd" of 15 to the annual meeting of 1884. The total potential was but a microscopic speck to the vast live stock industry of the nation.

Building a Profession

During the 15 years following 1885, a veterinary profession was built. Successful graduates of the short-term schools or of the none-too-long-term public ones inspired quite a deluge of students to follow suit. During that decade and a half, the schools increased their courses of instruction from two to three years, matriculation examinations were started, more schools, private and public, were established, organized veterinary medicine began to attract support, and practice laws were passed in nearly every state. Fifteen years is a short period of time, one year shorter than the time since most of the private schools closed. In veterinary history, the period stands out in bold relief. It may well be studied intently as a guide for future action. What was the power behind this remarkable achievement? The answer is "personnel," the first personnel of the veterinary profession in this country. To catalog it is to write the *livre d'or* of the veterinary service.

Just what happened from 1885 to 1900? Veterinary schools had flourished in foreign countries since the sixth decennium of the eighteenth century, and they had created a profession of veterinary medicine. The United States Veterinary Medical Association (A. V. M. A.) was the watchful guardian of the march in this country. Its meetings were characterized by heated discussions over the educational standards which were doggedly making a profession where none existed before. Common sense always won. If the educational quality of the graduates was not high, it was not only consonant to the career the forming profes-

sion had to offer, but was comparable to that of contemporaneous professions. Physicians were still being made in two years; lawyers "read law" at the local attorney's office; dentists qualified by working with others; and "hell-fire" preachers came from hither and yon duly ordained to save one's soul. Moreover, high school graduates were not numerous, least of all among the young men an unmade profession could hope to attract. What association of the nineteenth century could have lured high school graduates into an untried, adolescent undertaking that had been degraded by quackery for more than a century of American independence—an undertaking that was still fighting for a semblance of legal recognition with the chance of winning manifestly uncertain?

Not Particularly Inviting

So, the achievements of the 15-year period above mentioned was not an easy task nor an idle one, as time has shown. To have leaped to higher standards for a practically non-existent profession would have been a poor type of strategy in the laborious work of building up a classical veterinary service for this country. The forming profession had little but vague hope as an enticement. There was nothing of a substantial nature to offer the few young men of the period fortunate enough to have acquired a high school or college education. But let it not be forgotten, however, that the primary education of the period included the more essential high school subjects. The average matriculants of the veterinary schools were not ignoramuses by any means. Elementary geometry, algebra and physical geography were not foreign to the scholars of the "little red school house." Many of the veterinary students made better use of basic grammar than some of the conventionally crammed of the present time, and among them were graduates of high schools, normal schools and academies, school teachers and a smattering of finished products with two-letter decorations.

That certain schools should never have been founded is incontrovertible, nor is it deniable that incompetents did not creep in. But, the fact remains that a profession was built through an indomitable leadership that kept both radical and reactionary groups in check in the matter of soaring too high and sinking too low in the educational field—the source of supply for the indispensable personnel. Vague as the issue may seem to the casual, it remains extant today more intensified than during the formative stage. "My kingdom for a personnel!" remains and always will remain the principle issue of any profession.

Unsolved Problems

If veterinary service is not done by specially trained men, others will do it regardless of the consequences. What the consequences will be is conjecture. But, organized veterinary medicine believes the consequences will be grave. Rabies, Bang's disease, bovine mastitis, plant and forage poisonings, grave parasitisms, insect enemies run rampant. Poultry diseases are passing from professional hands. Local milk and meat inspection is yet to come. Classical veterinary medicine for every farm and home of the nation is a hope of the far-distant future. The work of the national association, however, is to guide the profession to that objective. Its hopes lie in an efficient personnel of sufficient magnitude, as much at the present time as in the formative period.

What occurred during the first 20 years of the twentieth century was merely the progress running parallel to the other professions. During this period there came the World War, the replacement of horses to a considerable extent by motor power, and the closing of the private schools. Deterrents? No. The World War gave the army an excellent veterinary corps. The replacement of horses developed neglected bovine, porcine, ovine and avian medicine to a higher level. The closing of the private schools improved veterinary education and brought prestige to veterinary science. In all of these, within its power, the national association played a major rôle, but never yielded the fundamental principle of furnishing the American people a sufficient number of reasonably well qualified veterinarians as its first obligation.

Insufficient Support of Veterinary Colleges

So, at the present time, when the schools to which the responsibility of furnishing a personnel for the veterinary profession has been delegated, are restricting entrants to comply with the limited accommodations and facilities that state legislatures are supplying, the time is not opportune for placing the struggling schools in an embarrassing position. When state legislatures make their meager appropriations for veterinary colleges, are they governed by the needs of the live stock industry of the nation? Obviously, they give little thought to the needs beyond state boundaries and perhaps too little to those within. So, again, the national association rules that the personnel of the veterinary profession should not be ruthlessly curtailed. Those who declare that restricting enrollment in the veterinary schools is quite a coup should consider that for the tremendous live stock

industry developed in the United States and Canada, all of the veterinary colleges from the beginning have furnished only a meager 20,000 veterinarians and that at the present time the survivors are dying off faster than recruits are allowed to enter, notwithstanding the growth of animal production and its ever-increasing disease problems.

The above, this writer believes, explains the action of the House of Representatives at Columbus on the grading of veterinary colleges at this moment. The national association has never overlooked public welfare, and it has always been the champion of higher standards.

Ohio Short Course for Milk Inspectors

A short course for milk inspectors will be held by the Department of Dairy Technology, College of Agriculture, Ohio State University, March 1-2-3, 1937. A similar course was given last year and ten veterinarians were among those in attendance.

Value of Veterinary Service Emphasized

Dr. J. P. Foster, of Minneapolis, Minn., has contributed three articles, "The Management of Brood Mares," "Bog Spavin" and "Umbilical Hernia," to *The Thoroughbred Record.* These appeared in the September, November and December, 1936, issues. In the discussion of each subject, the importance of employing competent veterinary service was stressed by the author.

Milk Ordinance

Veterinarians who are interested in regulating the distribution of milk and cream by ordinance, will find that the one adopted by the Board of Health of Steubenville, Ohio, is a useful guide. Among provisions of the ordinance is one requiring that certificates showing the herds to be free from tuberculosis must be issued by qualified veterinarians. The ordinance specifies that after January 1, 1938, all milk and cream sold in Steubenville must come from herds that are free from Bang's disease. Certificates showing freedom from Bang's disease must be issued by veterinarians.

A mind, once formed, is never suited after; one yet in growth will ever grateful be.—Goethe.

ROARING IN A FOAL*

By E. C. W. SCHUBEL *and* J. C. SCHWABLAND
Blissfield, Mich.

A foal which showed difficulty in breathing at birth was born on July 31, 1935. Two days later, we were called by the owner to determine the cause of the trouble. There seemed to be a bronchitis present, as auscultation revealed râles in the bronchi. Various expectorants were used during the following six months to clear the respiratory tract of the mucus. There were periods when the colt would breathe more easily, but the trouble did not disappear. When the colt was about six months old, further treatment was discontinued.

At the clinic of the Michigan-Ohio Veterinary Medical Association, held in Blissfield, on May 14, 1936, more than 50 veterinarians made observations on the colt. Diagnoses of heaves, bronchitis and collection of the guttural pouches were made. On June 23, the colt was taken to the clinic held by the Michigan State Veterinary Medical Association, in East Lansing. To determine the location of the cause of the labored breathing, a tracheotomy was performed by Dr. F. E. Stiles, of Battle Creek. When the tube was inserted into the trachea, the colt breathed more easily. It was then believed that the colt was a roarer. Dr. T. A. Sigler, of Greencastle, Ind., operated on the colt. He found, upon opening the larynx, that there was a bilateral paralysis. After recovery from the operation, the colt did not show much distress in breathing.

According to a large number of veterinarians present at the clinics, roaring in colts had not been observed in their experience.

When the colt was examined in October, it seemed to have recovered from the distressing symptoms of difficult breathing which were present for over ten months.

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OBSERVATIONS ON ANAPLASMOSIS IN IDAHO*

By GEORGE W. STILES, JR., *Denver, Colo.*
Pathological Division, Bureau of Animal Industry
United States Department of Agriculture

During the summer of 1936, the writer's attention was directed to cattle losses in southeastern Idaho, the cause of which had not been determined.

The disease in question appeared in only one herd of adult grade Herefords. The owner of the cattle had lived in that vicinity several years and never before had experienced cattle losses of a similar nature. Although the history and symptoms suggested anaplasmosis, the fact that that disease had not hitherto been diagnosed in the state caused some question among the local veterinarians and state officials as to the exact nature of the malady.

History of the outbreak: In a herd of 82 adult animals, 15 became sick and six died after an illness of a few days. The animals died on the following dates: June 17, June 20, June 21, June 28, August 8 and September 7.

The sick cows, which eventually recovered, became ill during the month of June. The ages of the cows that died and those that recovered are shown in table I.

TABLE I—*Ages of cows that died and recovered.*

AGE	DIED	RECOVERED
1 year	0	1
2 years	0	2
3 years	2	3
4 years	1	0
5 years	2	2
8 years	1	1
Totals	6	9

Symptoms reported: All affected animals became pale, anemic and jaundiced. They were observed to eat dirt and stones, indicating a depraved appetite. The milk secretion was impaired, and afflicted animals became thin and emaciated. Bloody urine was not observed. Recovery was slow.

Field and laboratory investigations: On September 24, the writer visited this herd and made blood-smears from five animals, reported by the owner as having been sick, and from two

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of them drew about 50 cc of blood under aseptic conditions. These samples were defibrinated and saved for inoculation purposes. Microscopic examination of the stained blood-smears revealed the presence of marginal bodies in all five cases. The anaplasms were few in number.

Inoculation experiments: The blood samples were chilled and forwarded for inoculation purposes. One sample was sent to the Pathological Laboratory, U. S. Bureau of Animal Industry, Washington, D. C., which, when inoculated into a susceptible cow, produced a typical case of anaplasmosis after 42 days, rather a longer period of incubation than usual. This animal recovered.

The second blood sample was injected into cow 192, on October 1, by Dr. Lewis H. Moe, station veterinarian, Oklahoma Agricultural Experiment Station, Stillwater, Okla. The first marginal bodies appeared October 19, and the cow died November 3, of anaplasmosis. Prior to death, her blood showed about 35 per cent of the red cells infested with anaplasms.

Possible source of infection: According to local, state and federal veterinarians, anaplasmosis has not been recognized heretofore in the state of Idaho. However, this disease is known to exist in the adjoining states of Oregon, Nevada and Wyoming.

The owner of the infected Idaho herd states there have been no new cattle introduced into his herd for the past three years. However, one of his cows died during the summer of 1934 from an unknown cause.

There are several possible means of introducing this disease into new localities. Perhaps the "carrier" animal is the most likely source of infection. Infected stray animals might be responsible for spreading anaplasmosis. A large herd of cattle showing symptoms of "jaundice" and some death loss was reported grazing over this mountainous area during the past summer. Also, in this sage-brush district, myriads of ticks, especially *Dermacentor andersoni*, were reported during the spring and early summer months prior to the onset of this outbreak. Unpublished data from the work of Sanborn, Moe, and the writer* have definitely shown that interrupted feeding of both male and female adult *D. andersoni* ticks can transmit anaplasmosis from sick to healthy animals. Rees[1] has shown that, under controlled experimental conditions, this tick is capable of transmitting the disease. Very few horse flies were reported during the summer in this locality where the Idaho outbreak of anaplasmosis occurred. It appears probable that anaplasmosis may have existed

*The anaplasmosis project under federal and state coöperative agreement is being conducted by Prof. C. E. Sanborn, entomologist, Dr. Lewis H. Moe, veterinarian, and the writer, at the Oklahoma Agricultural Experiment Station, Stillwater, Okla.

in Idaho during the past summer, and it could be presumed that some carrier animal in that vicinity might have been responsible for the source of the infection. The exact mode of transmission of the infection in this outbreak was not definitely determined.

Anaplasmosis has been diagnosed and reported from the following states in the United States:

Alabama	Florida	Missouri
Arizona	Georgia	Nevada
Arkansas	Idaho	Oklahoma
California	Kansas	Oregon
Colorado	Louisiana	Texas
Delaware	Maryland	Wyoming
	Mississippi	

SUMMARY

Anaplasmosis is reported from the state of Idaho. The diagnosis was made by field investigations and microscopic examination of blood-smears, and was confirmed by animal inoculations.

The disease, anaplasmosis, has been observed and reported from 19 states in the United States.

ACKNOWLEDGMENTS

Appreciation for services rendered and courtesies shown are extended to Dr. D. E. Madsen, pathologist and veterinarian, Logan, Utah; to Dr. T. W. White, state veterinarian of Idaho, and to Dr. S. E. Nelson, practitioner, Preston, Idaho.

REFERENCE

[1]Rees, C. W.: The experimental transmission of anaplasmosis by *Dermacentor andersoni*. Parasitol., xxiv (1933), pp. 569-575.

Untested Cattle Cause Persistence of Bang's Disease

One of the principal reasons why Bang's disease persists in certain herds of cattle, even after several tests and the removal of reactors, is the addition of untested animals to these herds, according to reports by veterinary officials of the U. S. Bureau of Animal Industry, who have made an analysis of the reports on Bang's disease work in nine states.

Officials believe that the loss of cattle can be reduced materially if veterinarians will advise their clients how to handle their replacements. Purchases should be restricted to so-called negative herds or cattle which have passed the test, and which have been held in quarantine until a second negative test is made.

PRACTICAL VETERINARY PHARMACOLOGY, MATERIA MEDICA AND THERAPEUTICS. Howard J. Milks, Professor of Therapeutics and Director of the Small-Animal Clinic, New York State Veterinary College at Cornell University. 3rd edition. 581 pages, with 33 illustrations. Alexander Eger, Chicago, 1936. Cloth, $6.00.

The report of the A. V. M. A. Committee on Proprietary Pharmaceuticals, made at the 1936 meeting, directed attention to the fact that veterinary medicine had been recognized for the first time in the sixth edition of the National Formulary. Drugs and preparations used extensively by veterinarians, with dosages for different animals, were included.

It has been six years since the publication of the second edition of Dr. Milks' well-known text-book. The third edition has been made to conform with the U. S. Pharmacopoeia and to include those drugs and preparations in the National Formulary VI used by veterinarians.

The text of the previous editions has been revised. New sections have been added on vitamins and insecticides and on the treatment for heartworm infestation.

In the section on heartworms are mentioned fuadin and filsol, which have been recommended for combating these parasites. The actions of the drugs, their contra-indications and methods of administration, as well as reactions to the drugs, are fully explained. The medication of chickens infested with various parasites is outlined in the chapter on specifics for the alimentary tract.

The chapter on drugs acting upon the respiratory system has been enlarged to include lobeline, cocaine, carbon dioxide, metrazole and coramine as stimulating agents to respiration. The section on expectorants has been revised and their actions more fully explained. Their use is given in a table outlining which drug should be the most applicable to each kind of cough.

In the section on nerve depressants, the barbitals have been given much attention. The sections on toxicology of prussic acid, the salicylates, local anesthetics and preparations of the

pituitary body have been revised. Among the newer agents included in the book are: allantoin, alumnol, amytal sodium, artificial maggots, avertin, benzedrine, carotene, coramine, evipal, filsol, fuadin, liver extracts, several iodine preparations, halibut-liver oil, merthiolate, nupercaine, pentobarbital sodium, pentonucleotide, pine oil disinfectants, pitressin, pitocin, pyrethrin, rotenone and several of the salicylates.

The section on aniline dyes describes some of these agents which are used in medicine and more particularly those used as antiseptics.

The chapter on veterinary biologics has been revised. It gives information on allergy, anaphylaxis, nonspecific therapy, and outlines the use of biologics in some of the infectious diseases. Diagnostic products and their uses are fully described. The index is well arranged, and an excellent index to prescriptions is included. The aim has been not only to give a concise description of the action of each agent but to couple this action with the use of the drug in the treatment of disease. The practicing veterinarian will find the book very helpful in solving some of the puzzling problems he frequently encounters.

CLINICAL DIAGNOSTICS OF THE INTERNAL DISEASES OF DOMESTIC ANIMALS. B. Malkmus, former Professor of Pathology and Director of the Veterinary High School, Hannover, Germany. 11th edition. 302 pages, with 72 illustrations. Alexander Eger, Chicago, 1936. Cloth, $3.50.

The new edition of this standard text-book reflects the great progress that has been made in the diagnosis of internal diseases of animals, since the first English translation of this work appeared. Many parts of the previous editions have been entirely rewritten, especially the chapter on blood.

Because of its importance in the diagnosis and prognosis of animal diseases, the chapter on blood has been enlarged to include a discussion not only of color, coagulability and specific gravity, but the determination of blood groups, microscopical examination of blood, pathology of erythrocytes, pathology of the white blood picture, and many other phases of hematology. The changes in the blood picture caused by glanders, influenza, strangles, canine distemper, infectious anemia, tuberculosis and sporadic diseases are outlined.

The revision and translation from the eleventh German edition was under the direction of Drs. John R. Mohler and Adolph Eichhorn. As an indication of the wide acceptance of this work,

it may be mentioned that this edition has been translated also into Italian and Spanish.

The practicing veterinarian as well as the veterinary student will find this book a valuable source of information on the very latest diagnostic methods. The author has pointed out that diagnosis is the most difficult part of veterinary medicine and that everything depends on a correct recognition of the disease.

Hog Flu

According to warnings issued by Dr. J. L. Axby, State Veterinarian of Indiana, and Dr. H. C. Rinehart, Chief Veterinarian, Illinois Department of Agriculture, it would appear that a widespread infection of hog flu may exist in these states. Veterinarians have been advised to take the same precautions in handling this disease as they would in an outbreak of hog cholera.

Veterinarian Goes in for Arabian Horses

Dr. Urton Munn, practitioner of Athens, Ala., is interested in pure-bred Arabian horses. The accompanying photograph shows him astride his filly, Gasara, 14:3 hands high, weight 900 pounds. Dr. Munn also owns three other Arabians, Kaibab, Al Azhar and Pera.

Doctor Munn on His Favorite Mount

STUDIES ON NEUROTROPIC RIFT VALLEY FEVER VIRUS: THE SUSCEPTIBILITY OF SHEEP AND MONKEYS. G. M. Findlay, R. D. MacKenzie and Ruby O. Stern. Brit. Jour. Exp. Path., xvii (1936), p. 431.

The neurotropic strain of Rift Valley fever (an epizoötic found in 1930 in flocks of sheep and lambs pastured on the shores of Lake Naivasha in Kenya, British East Africa) virus produces encephalitis when introduced intracerebrally in lambs. Subcutaneous inoculation into sheep and lambs does not cause illness. Immunity against the pantropic strain follows the subcutaneous injection of neurotropic virus in sheep and lambs. Two species of monkeys, *Macaca mulatta* (the rhesus monkey) and *M. irus* (the crab-eating macacque), are highly susceptible to intracerebral inoculation of neurotropic Rift Valley fever virus and die with encephalitis. With an increase in the number of mouse passages there is a greater tendency for the virus, when injected intracerebrally in monkeys, to be found exclusively in nerve tissues.

STUDIES ON NEUROTROPIC RIFT VALLEY FEVER VIRUS: "SPONTANEOUS" ENCEPHALOMYELITIS IN MICE. G. M. Findlay and R. D. MacKenzie. Brit. Jour. Exp. Path., xvii (1936), p. 441.

Two cases of spontaneous encephalitis in mice are recorded following intraperitoneal inoculation of pantropic Rift Valley fever virus. The viruses isolated from the brains of these two mice did not show enhanced neurotropism. Both the mice developing spontaneous encephalitis already possessed a high degree of visceral resistance to infection with Rift Valley fever virus. In mice possessing a high degree of visceral resistance to infection with Rift Valley fever virus, intracerebral inoculation or intranasal instillation of pantropic virus may lead to the development of encephalitis. In such cases of encephalitis the presence of virus in the brain may be readily demonstrated, demonstrated only on dilution, or undemonstrable.

THE EFFECT OF HEMOLYTIC SUBSTANCES ON WHITE CELL RESPIRATION. Eric Ponder and John McCleod. Jour. Gen. Physiol., xx (1936), p. 267.

Substances such as saponin, the bile salts, etc., which produce lysis of red cells, also produce cytolysis of white cells from rabbit peritoneal exudates, the arbitrary criterion of their cytolytic effect being their ability to depress the oxygen consumption of the leukocytes. The amount of cytolysis increases regularly as the amount of the added lysin is increased and sufficiently large quantities of saponin, sodium taurocholate, sodium glycocholate or sodium oleate are capable of virtually abolishing the oxygen consumption altogether. At the same time, it can be shown that a lysin such as saponin is used up in combining with the white cells and the reduction in oxygen consumption appears to be roughly proportional to the amount so combined. The action of these lytic substances on white cells, in fact, is very similar to their action on red cells, due allowance being made for the fact that the cytolysis of the white cell is probably not an all-or-none process like hemolysis. White cell respiration is also depressed in hypotonic solutions, the respiration being virtually linear with the tonicity.

THE NECESSITY OF LIVING CELLS FOR THE CULTIVATION OF PSITTACOSIS VIRUS. F. O. MacCallum. Brit. Jour. Exp. Med., xvii (1936), p. 472.

The results of these experiments support the view that the virus of psittacosis is an obligate intracellular parasite. The "elementary bodies" and "large forms" failed to reproduce in a cell-free medium. Attempts to infect tissue cultures with virus in the "large form" have invariably failed, presumably because the large forms find it difficult to enter cells.

TISSUE CULTURE STUDIES ON BACTERIAL HYPERSENSITIVITY. III. The persistence *in vitro* of the inherent sensitivity to tuberculin of cells from tuberculous animals. Johannes K. Moen. Jour. Exp. Med., lxiv (1936), p. 943.

Suspensions of scattered mononuclear cells were obtained from tuberculous guinea pigs by the intrapleural injection of warm molten parowax. Such cells showed marked sensitivity to the toxic action of tuberculin when tested in tissue culture. Convincing evidence is offered that sensitivity to tuberculin is an inherent characteristic of cells from tuberculous animals. The

transformation of mononuclear exudative cells into fibroblastic growth was demonstrated and showed persistence of sensitivity to the action of tuberculin on repeated transplantation over a prolonged period *in vitro*. There is no proof that tuberculin allergy is the result of an antigen-antibody reaction. The nature of the toxic action of tuberculin on cells from tuberculous animals when tested *in vitro* is a matter of conjecture.

EFFECT OF PNEUMOCOCCUS AUTOLYSATE ANTITOXIN ON PNEUMOCOCCUS PNEUMONIA IN GUINEA PIGS. Julia T. Weld and Helena Gilder. Jour. Exp. Med., lxiv (1936), p. 953.

In rabbits, guinea pigs and mice, pneumococcus autolysate antitoxin has no effect against generalized infection by the pneumococcus. In a large proportion of cases of pneumonia and sepsis caused by the intratracheal inoculation of large amounts of living pneumococci in normal and sensitized guinea pigs, autolysate antitoxin saves the animal. The autolysate antitoxin is protective in most cases of pneumonia and sepsis caused by the intratracheal inoculation of small amounts of living pneumonia combined with sublethal doses of staphylococcus toxin. The experiments indicate that in pneumococcus pneumonia of guinea pigs the autolysate toxin or toxins are elaborated only in the lungs.

THE LIFE HISTORY OF PSOROPTES COMMUNIS, VAR. OVIS WITH PARTICULAR REFERENCE TO LATENT OR SUPPRESSED SCAB. W. Downing. Jour. Comp. Path. & Therap., xlix (1936), p. 183.

The life cycle of *Psoroptes communis* var. *ovis* is usually completed in eleven days under climatic conditions of Great Britain with a minimum period of ten days and a maximum period of twelve days. The number of eggs laid by ovigerous females show relatively little individual variation, but the rate of oviposition shows considerable variation. This variation appears to be dependent upon temperature, humidity and skin reactions inimical to the parasite. Sheep heavily infected in winter recover spontaneously in the summer. This recovery may be complete or more frequently followed by a relapse in the following autumn, the scab parasites having remained latent during the summer. Acari are continuously present throughout the latent period, whereas in recovery the acari fail to survive the complete latent period. When infection is contracted in the spring or summer, the scab may remain active but progress is very slow until autumn. Some

sheep harbor a few acari in the summer without showing any clinical symptoms of the disease. The infraorbital fossae, the inguinal folds, the scrotum of the wether and the tail are important sites for latent scab. In the autumn, migration occurs to the general body surface. Healthy flocks may be infected by sheep in the summer which are apparently clean but on which scab is latent. The most favorable time of the year to dip sheep for control of scab is the autumn, when migration from the sheltered local sites to the general skin surface of the body occurs.

THE PROTEIN CHANGES IN OX SERUM INDUCED BY HYPERIMMUNIZATION AGAINST RINDERPEST VIRUS. M. H. French. Jour. Comp. Path. & Therap., xlix (1936), p. 226.

An increase of total protein, total globulin, euglobulin and globulin insoluble in saturated sodium chloride solution takes place as a result of intramuscular hyperimmunization of cattle against rinderpest virus. The changes in the serum proteins of cattle given a massive dose of virulent blood intrarumenally can be as high as those for cattle hyperimmunized intramuscularly or subcutaneously. When the aqueous extract of the pulped virus containing organs is given intrarumenally, the serum protein changes can be as high as those obtained with intramuscularly hyperimmunized cattle. Hyperimmunization may bring about an increase of the pseudoglobulin proportions as well as the normal increase of euglobulin. Antisera made by the intrarumenal method are inferior to those prepared by the more established technic.

A BRUCELLA ABORTUS SUSPENSION OF UNIFORM AGGLUTINABILITY STANDARDIZED BY MEANS OF A DRY STABLE STANDARD ANTI-ABORTUS SERUM. A. W. Stableforth. Jour. Comp. Path. & Therap., xlix (1936), p. 251.

The technic of preparing a dry standard antiabortion serum by drying the serum of a cow naturally infected with *Brucella abortus* to constant weight over phosphorus pentoxide at low pressure is explained. The antibody content of the serum was slightly lowered by drying but has remained constant 2½ years after drying. The dried antiabortion serum is used to obtain suspensions of *Br. abortus* of uniform agglutinability, standardization being accomplished by titration. Thirty-seven batches of suspensions have been prepared in 2½ years by the same method from the same strain and little difficulty was encountered in

maintaining the standard agglutinability, 25 per cent agglutination with a 1:480 dilution of the reconstructed dried serum. Adjustment of the agglutinability has been made by slight alteration of the opacity of the suspension. Suspensions keep well at room temperature or in the cold. Some loss of agglutinability occurs during the first one or two weeks but suspensions issued three or four weeks after washing off can be relied upon to maintain their agglutinability unaltered for three months or longer.

THE SERUM PROTEIN CHANGES DURING THE IMMUNIZATION OF GOATS AGAINST RINDERPEST VIRUS. M. H. French. Jour. Comp. Path. & Therap., xlix (1936), p. 246.

The serum protein changes during immunization of a goat against rinderpest virus are chiefly an increase of serum globulins similar to that occurring in the immunization of the ox. In the goat the increase in pseudoglobulin is much more than is found in the ox, while the reverse is true for the euglobulin fraction. The indications are that the antiviral body is spread over a wider serum-protein range in the goat than in the ox.

STUDIES ON INCUBATOR HYGIENE. VI. A note on the resistance of *Salmonella pullorum* on sterilized and unsterilized egg shell. Robert Graham and V. M. Michael. Poultry Sci., xv (1936), p. 490.

Very little variance in the longevity of *Salmonella pullorum* on egg shells which had been sterilized at 15 pounds steam pressure for 30 minutes and those which had not been sterilized previous to contamination with *S. pullorum* was observed following three separate formaldehyde fumigations of a forced-draft incubator. *S. pullorum* on egg shells in hatching trays survived formaldehyde fumigation for 30 minutes. After 40 minutes, *S. pullorum* was generally non-viable, although in one fumigation it survived for 100 minutes on sterilized egg shells. The survival of *S. pullorum* on egg shells during formaldehyde fumigation of a forced-draft incubator for 100 minutes suggests the advisability of keeping the incubator closed for at least two hours following fumigation. The range of viability of *S. pullorum* in empty incubators is comparable to results obtained in the case of a 32,000-capacity machine when loaded to various capacities. In 25 separate fumigations, *S. pullorum* that occasionally survived 40-minute fumigations may have had a range of 100 minutes.

STUDIES ON INCUBATOR HYGIENE. VII. Results of fumigation on *Salmonella pullorum* in incubators at full capacity. Robert Graham and V. M. Michael. Poultry Sci., xv (1936), p. 492.

Formaldehyde fumigation is probably as effective in destroying *Salmonella pullorum* in a forced-draft incubator operated at 50 or 66 per cent egg capacity as in an incubator that is practically empty, since the average survivability of *S. pullorum* and range survivability proved comparable. In one instance *S. pullorum* survived for 150 minutes in the lower door and 120 minutes in the upper door and proved non-viable at 180 minutes and 150 minutes, respectively.

STUDIES ON INCUBATOR HYGIENE. VIII. Resistance of *Salmonella aertrycke, Salmonella anatum* and *Salmonella* from quail to formaldehyde fumigation. Robert Graham and V. M. Michael. Poultry Sci., xv (1936), p. 494.

Salmonella aertrycke and *Salmonella newport* on cotton plugs survived formaldehyde fumigation in forced-draft incubators for 30 minutes but were non-viable at 60 minutes, while *Salmonella* sp. isolated from baby quail survived for 60 minutes but were non-viable at 90 minutes.

PUBLICATIONS RECEIVED

A Consideration of the Accredited Producers' Scheme of the Milk Marketing Board. D. S. Rabagliati. Paper presented at the 54th Annual Congress, N. V. M. A. of Great Britain and Ireland, Sept. 14-18, 1936. pp. 8.

Some Observations on the Use of Vaccines in the Protection of Cattle Against Tuberculosis. R. E. Glover. Paper presented at the 54th Annual Congress, N. V. M. A. of Great Britain and Ireland, Sept. 14-18, 1936. pp. 8.

Enzoötic Abortion in Ewes. J. R. Greig. Paper presented at the 54th Annual Congress, N. V. M. A. of Great Britain and Ireland, Sept. 14-18, 1936. pp. 4.

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Kind Lady: "And how would you like a nice chop?"

Weary Tramp: "Dat all depends lady—is it lamb, pork or wood?"

—*Milk Inspector.*

Regular Army

Each of the following-named officers of the Veterinary Corps is relieved from further assignment and duty at the Army Veterinary School, Army Medical Center, Washington, D. C., effective upon completion of his present course of instruction, on or about December 30, 1936, and will proceed to the station specified after his name and report for duty:

1st Lieut. James B. Nichols, Fort Myer, Va.
1st Lieut. John H. Rust, 3d, Fort Benning, Ga.
1st Lieut. Bernard F. Trum, Fort Oglethorpe, Ga.

1st Lieut. Daniel S. Stevenson is relieved from his present assignment as a student at the Army Veterinary School and from further duty at Washington, D. C., effective on or about December 30, 1936, and is then assigned to duty in the Philippine Department. He will proceed to the New York Port of Embarkation, Brooklyn, N. Y., and report to the commanding general for duty until such time as will enable him to sail on or about March 18, 1937, for the Philippine Department, and upon arrival will report to the commanding general for duty with the Veterinary Corps.

Major Gardiner B. Jones is relieved from duty at the Presidio of San Francisco, Calif., effective at such time as will enable him to sail on the transport scheduled to leave San Francisco, Calif., on or about April 9, 1937, for the Philippine Department, and upon arrival will report to the commanding general for assignment to duty with the Veterinary Corps.

Major William H. Dean is assigned to duty at the Presidio of San Francisco, Calif., effective upon completion of his present tour of foreign service in China.

Major James E. Noonan is assigned to duty at Fort Belvoir, Va., effective upon completion of his present tour of foreign service in the Philippine Department.

The appointment of the following-named first lieutenants of the Veterinary Corps Reserve as first lieutenants in the Veterinary Corps, Regular Army, with rank from November 30, 1936, and their assignment to stations as indicated after their names, are announced:

Fred Lewis Herring, United States Military Academy, West Point, N. Y.
Wayne Devere Shipley, Fort Reno, Okla.
Don L. Deane, Fort Bliss, Texas.
Benjamin Francis Leach, Fort Sheridan, Ill.
Walter Smit, Fort Riley, Kan.

Lieutenants Shipley and Smit, now on duty with the Civilian Conservation Corps, will proceed in compliance with orders to be issued by their respective corps area commanders to the stations to which assigned, and report for duty. Lieut. Herring will proceed from Schuyl-

kill Haven, Pa.; Lieut. Deane from Huntington Park, Calif.; and Lieut. Leach from Detroit, Mich.; each to the station to which assigned and report for duty.

1st Lieut. Andrew J. Sirilo is relieved from further assignment and duty at the Army Veterinary School, Army Medical Center, Washington, D. C., effective upon completion of his present course of instruction, on or about December 30, 1936, is then assigned to station at Fort Mason, Calif., and will proceed to Brooklyn, N. Y., and report to the commanding general, New York Port of Embarkation, for temporary duty until such time as will enable him to sail on the transport scheduled to leave that port on or about March 18, 1937, for San Francisco, Calif.; upon arrival in San Francisco will report to the commanding general, San Francisco port of embarkation, for duty.

Veterinary Reserve Corps

NEW ACCEPTANCES

Miller, James Julius.....1st Lt...1371 Fulton St., San Francisco, Calif.
Phifer, Wm. Gough.....1st Lt...1617 11th St., Tuscaloosa, Ala.
Taylor, Clarence Edward....1st Lt...2614 Harney St., Vancouver, Wash.
Upchurch, John Wright.....1st Lt...716 N. Edgefield St., Dallas, Texas.
Walker, Wm. Kenneth.....1st Lt...Box 1086, Sacramento, Calif.

PROMOTIONS

To

Anderson, Horace L.....Major...Rockwell, Iowa.
Mayfield, Orley John.....1st Lt...304 3rd Ave., Charles City, Iowa.
Waddell, Wm. Fernando.....1st Lt...Box 708, Shelton, Wash.
Wempe, Lillis Raphael.....1st Lt...Seneca, Kan.
Wilder, Clifford W.....1st Lt...RFD No. 1, Chatham, N. Y.
Williamson, Wm. Welton....1st Lt...Barada, Neb.

SEPARATIONS

Leach, Benjamin F.....1st Lt...Accepted appointment in V. C., Regular Army.
Shipley, Wayne D.....1st Lt...Accepted appointment in V. C., Regular Army.
Smit, Walter .....1st Lt...Accepted appointment in V. C., Regular Army.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Altaker, Robert Roben.....1st Lt...Hq. 3rd Corps Area, Baltimore, Md.
Fisherman, Henry .....1st Lt...Fort Worth Dist., Fort Worth, Texas.
Foote, Bernard E.....1st Lt...Silver City Dist., Silver City, N. Mex.
Smith, Ivan .....1st Lt...Fort Snelling, Minn.
Todd, Guy Harold.....1st Lt...Fort Snelling, Minn.
Lassen, Keith O. .....1st Lt...Fort Benning, Ga.
Sullivan, Wm. G. .....1st Lt...Fort Bragg, N. C.

TERMINATION OF ASSIGNMENTS TO ACTIVE DUTY

Bartlett, Densil C. .....1st Lt...Hq. Dist. No. 4, CCC, Richmond, Va.
Burkey, Fred M. .....Capt...Lufkin Dist., Lufkin, Texas.
Siemer, Everett J. .....1st Lt...Silver City Dist., Silver City, N. Mex.
Shipley, Wayne Devere.....1st Lt...Fort Benning, Ga.
Smit, Walter .....1st Lt...Fort Leavenworth, Kan.

NEBRASKA STATE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Nebraska State Veterinary Medical Association was held at the Hotel Lincoln, Lincoln, on December 8-9, 1936. After the formal opening and invocation, the regular order of business was taken up.

At 11:05 o'clock, as per previous arrangement, the proceedings of the meeting were abruptly halted to take advantage of a five-minute radio broadcast under the caption of "Foundation of Animal Health." It was the initial number of a campaign of publicity sponsored by the Associated Serum Producers, Inc. Provision had been made for the broadcast to be heard in the convention hall. It came in perfectly. The address was made by Col. Robert J. Foster, president of the American Veterinary Medical Association. This arrangement was highly satisfactory to the members.

At 1:30 o'clock, the afternoon session was opened with a very practical talk by Dr. S. S. Gibson, of Randolph, on diseases of swine. The subject was liberally discussed by Dr. J. D. Ray, of Omaha, and others. Many points of value to the practitioner were brought out in the discussion.

Dr. W. L. Boyd, of the University of Minnesota, presented the subject of cattle diseases, with especial attention to Bang's disease and the relation that the disease bears to sterility. Dr. Boyd's classic presentation, supplemented by stereopticon views of the genital organs depicting various pathological conditions, was most interesting. The interest was augmented by the discussion led by Dr. O. E. Reihart, of Omaha. Many suggestions were made as to the handling of infected herds and individual animals in efforts to eradicate the disease. Discussion of the economic loss occasioned by Bang's disease was discussed very extensively.

Diseases of sheep were discussed by Dr. D. F. Luckey, former state veterinarian of Missouri, in his usual lucid manner. Dr. Luckey's extensive experience with sheep and sheep diseases enabled him to cover a wide field, taking up most of the troubles in sheep commonly met by the average practitioner.

The second day the meeting was opened at 9:00 o'clock with a discussion of small-animal practice by Dr. E. J. Frick, of Kansas State College. His discourse was most interesting and instructive and was especially appealing to the small-animal practitioner.

Col. Robert J. Foster, whose address had been postponed from the previous afternoon, then gave his talk on the subject of the American Veterinary Medical Association. Col. Foster made it plain that professional people should be associated for mutual gain and that there is work for a national organization as well as for local and state organizations. He called attention to the widening field for service by veterinarians and pointed out that they are essential in guarding and preserving public health. He stressed the thought that veterinarians should familiarize themselves with such work and be qualified to render service in sanitary regulatory work whenever the opportunity is presented. In his closing remarks, Col. Foster's plea for membership in the national organization resulted in obtaining 15 applications.

Prof. W. J. Loeffel, of the Animal Husbandry Department, University of Nebraska, was the next on the program, speaking on the timely subject of "Feeds and the Feed Situation." This talk brought out a vast amount of information that was highly valued and well received by the veterinarians in attendance. Dr. C. R. Collins, Osceola, chairman of the Publicity Committee, made an extensive report on that subject. Dr. L. H. Crumbaugh, Craig, who is both attorney and veterinarian, gave a splendid talk on essentials and benefits of organization.

At the afternoon session, Dr. Adolph Eichhorn, of Pearl River, N. Y., delivered a masterly discourse on "Developments and Investigations in Virus Diseases." Interest did not lag during this hour. Dr. E. R. Frank, of Kansas State College, followed with a very practical talk, accompanied by X-ray slides, on subjects pertaining to equine practice.

Lt. Col. J. D. Derrick, Corps Area Veterinarian, Omaha, stressed the need of veterinarians qualifying for service in dairy and meat inspection and in sanitary work.

The election of officers resulted as follows: President, Dr. D. W. Hurst, Tecumseh; vice-president, Dr. L. J. Boulier, Cozad, and secretary-treasurer, Dr. Jno. D. Sprague (reëlected), David City. Grand Island was selected for the place of meeting in 1937. Dr. O. H. Person, of Wahoo, was chosen as delegate and Dr. Floyd Perrin, Lincoln, alternate to the A. V. M. A. House of Representatives for two years.

Dr. J. L. George, of Chester, and Dr. R. W. Johnson, of Lincoln, were elected to membership.

Registration for the two days was 161, including 22 non-members from adjoining states. At the banquet the evening of the first day, 176 plates were laid. Dr. L. Van Es acted as toastmaster. Responses were given by Col. Foster, Drs. Eichhorn, Dykstra and Peters and others. After a most enjoyable two hours, the floor was cleared for the usual dance.

The ladies, during each afternoon, were most graciously entertained by the auxiliary under the guidance of Mrs. Neal Plank, Mrs. F. Woodring and Mrs. E. H. Meyer, the local committee.

JNO. D. SPRAGUE, *Secretary-Treasurer.*

DELAWARE VETERINARY MEDICAL ASSOCIATION

Twenty-five veterinarians and friends gathered at noon in the Banquet Room of the Hob Tea Room, Delaware Trust Building, Wilmington, December 22, 1936, for the annual meeting of the Delaware Veterinary Medical Association. Dr. C. C. Palmer, President, presided at the meeting. After the gathering partook of a splendid turkey dinner, Dr. Palmer called the meeting to order and introduced Mayor W. W. Bacon, of Wilmington, who delivered the address of welcome. As indicated by his remarks, Mayor Bacon, who is a business man associated with the duPont company and an unusually efficient mayor, possesses a very clear understanding of the services rendered by the veterinary profession to the community. This fact is further indicated by Mayor Bacon's appointment of Dr. C. I. Hoch to the Wilmington Board of Health. The response to the address of welcome was delivered by the President, in which he eulogized the veterinarian and the veterinary profession.

The guest speaker was Dr. Harry M. Martin of the School of Veterinary Medicine of the University of Pennsylvania. Dr. Martin talked on parasites in the dog. This was a splendid address delivered in an interesting manner and contained much information of interest to the practicing veterinarian. The discussion following Dr. Martin's address was chiefly along lines of parasite control in the dog and considerable time was devoted to the discussion of heart worms, a parasitic disease quite prevalent in this part of the country.

Dr. R. M. Sarde, of the State Board of Agriculture, read a paper on Bang's disease, in which he presented the recommendations of the Committee on Bang's Disease of the United States Live Stock Sanitary Association. This report stimulated the usual discussion which is held at most veterinary meetings regarding Bang's disease.

One new member was added to the Association, Dr. Walter C. Fetherolf (U. P. '33), who is working part time as dairy inspector for a large milk distributor and part time in practice at Centerville.

Dr. R. M. Sarde was elected delegate to the House of Representatives of the A. V. M. A. and Dr. C. C. Palmer alternate for the two-year term starting January 1, 1937.

The following officers were elected for the ensuing year: President, Dr. Louis Levinson, Middletown; vice-president, Dr. Maurice L. Zurkow, Dover; secretary-treasurer, Dr. Harvey L. Fell, Wilmington.

C. C. PALMER, *Reporter.*

UNIVERSITY OF PENNSYLVANIA CONFERENCE OF VETERINARIANS

The thirty-seventh annual Conference of Veterinarians at the University of Pennsylvania was held January 5-6, 1937, at the Veterinary Department buildings in Philadelphia. The program committee, comprised of Drs. C. J. Marshall, Chairman, W. J. Lentz, H. M. Martin, W. J. Lee and M. A. Emmerson, had in its plans the desire to provide a diversified subject matter, to appeal to as many different interests as possible, yet with topics essentially of live interest to practitioners. As a matter of record, this motivation has been preserved from the beginning in these annual conferences. Dr. Leonard Pearson, who inaugurated the series in 1900, did so with this explicit object. The recent meeting has fittingly coördinated the "broader public aspect of veterinary science" with informed discussions on "everyday private practice."

The first session, Tuesday afternoon, was under the direction of Dean G. A. Dick. Dr. J. H. Penniman, Provost of the University, inaugurated the session with an address of welcome in which the importance of worthwhile research was stressed and its relationship to the practice of all the sciences. Dr. Penniman presented some interesting data on recent developments in the University in general, many of which applied particularly to the Veterinary Department. This was followed by a summary of the studies of "Coöperative Bang's Disease Work in the United States," by Dr. A. E. Wight, Chief, Tuberculosis Eradication Division, U. S. Bureau of Animal Industry, Washington, D. C. A paper was then presented dealing with "Gas Gangrene Infection in the Dog," under the combined authorship of Drs. J. A. S. Millar, of Deal, N. J., and W. P. Rawlings and W. G. Love, of

Swiftwater, Pa. In this very interesting and informative article the actual occurrence of *Clostridium welchii* (*Bacillus perfringens*) infections was described, together with adequate measures for prophylaxis and treatment. The afternoon session was brought to a close with a paper entitled, "The Prevention of Blackleg," by Dr. J. P. Scott, research associate at the University.

The evening session, under the direction of Dr. R. S. Amadon, consisted in a splendid address by Dr. Edward Lodholz, professor of physiology in the Graduate School of Medicine, entitled, "Fatigue in Domestic Animals and Man. Its Advantages, Efficiency and Dangers." In his extremely interesting style, Dr. Lodholz presented a most informative argument against both over-fatigue and succumbing to the effects of a mild degree of fatigue, which is a more common occurrence, particularly in the case of mental activity. This was followed by an illustrated lecture on the part of Drs. E. L. Stubbs and J. D. Beck, of the veterinary faculty, entitled, "European Reminiscences." It was somewhat in the nature of a travelogue, covering a number of the principal European veterinary schools, together with some delightful pictures of Switzerland. The latter were introduced, according to Dr. Stubbs, with the principal object of strengthening interest in the coming International Veterinary Congress of 1938.

On Wednesday, the morning session, under the direction of Dr. H. M. Martin, was introduced with a very scholarly paper on "The Diagnosis of Catarrhal Mastitis," by Dr. Louis A. Klein. This report was received with considerable interest by a large number of those present, and deservedly so. Both the natural interest in the subject, and the prominent position which Dr. Klein occupies in this field combined to establish this report as one of the most outstanding of the Conference. This was followed by the reading of a paper entitled, "Gastro-Intestinal Diseases of the Dog and Their Differentiation," by Dr. N. J. Pyle, of Pearl River, N. Y. In the unavoidable absence of Dr. Pyle, Dr. Adolph Eichhorn read and discussed the subject matter in a most satisfactory manner. The morning was brought to a close with a talk on "Breeding Problems in Horses," by Dr. F. A. Marshall, of Harrisburg, Pa. The speaker illustrated his talk by demonstrating several of the procedures carried out on some of the most progressive stud farms. The demonstration was most helpful and illustrated the lack of adequate knowledge in the management of one of the fundamental industries of our state.

Dr. E. L. Stubbs presided over the afternoon session of the same day. In the absence of two of the appointed speakers, Dr. M. F. Barnes, of the Pennsylvania B. A. I. laboratories, and Dr. H. M. Kalodner, State Veterinarian, reviewed the recent devel-

opments in the investigation and control of diseases under the supervision of the Pennsylvania Bureau of Animal Industry. Dr. F. R. Beaudette, of New Brunswick, N. J., supplied some very interesting observations on the recent developments and contemporaneous studies of fowl-pox in the state of New Jersey. Although not on the program, Dr. Beaudette's paper was judged one of the most interesting of the entire series. Finally, Dr. W. J. Lee, of the University staff, presented a demonstration in the court-yard on various methods of handling the different problems in the restraint of animals.

The fact that the attendance continues to increase year after year, and this year it was so great as, more than ever, to present a problem of seating the audiences in the crowded lecture-rooms, attests both to the far-sighted wisdom of Dr. Pearson and the satisfactory manner in which his aims have been carried out.

A. H. CRAIGE, *Reporter.*

NEW MEXICO VETERINARY MEDICAL ASSOCIATION

The semi-annual meeting of the New Mexico Veterinary Medical Association was held in Foster Hall, at the New Mexico A. & M. College, State College, January 8-9, 1936, with Dr. W. L. Black, of State College, presiding. The meeting was unusual in that it was held in two states, New Mexico and Texas, and one foreign country, Mexico.

After the invocation by Rev. H. D. Buchanan, of State College, the address of welcome was given by Dr. Ray Fife, president of the New Mexico A. & M. College. On account of illness in his family, Dr. S. W. Wiest, of Santa Fe, was prevented from attending the meeting, so Dr. F. L. Schneider, of Albuquerque, responded to the address of welcome.

The program for the first session consisted of the following subjects:

"Some Aspects of Rabies," by Dr. H. C. Schipman, Las Cruces.

"Some Poisonous Plants of the Southwest," by Dr. F. P. Mathews, Texas Agricultural Experiment Station, Alpine, Texas.

"Things Veterinarians Can Do to Assist Ranchmen," by A. D. Brownfield, president of the New Mexico Cattle Growers' Association, Florida.

At the afternoon session, Col. Robert J. Foster, president of the American Veterinary Medical Association, gave an interesting talk on the national organization and the benefits to be derived from membership in it. The subject, "Postmortem and Specimen Preservation," was discussed by Dr. H. E. Kemper, of Albuquerque.

Dr. Black gave his presidential address to conclude the afternoon session. The banquet was held in the evening at the New Tivoli Hotel, in Juarez, Mexico.

On the morning of the second day, a small-animal clinic was held in the hospital of Drs. Koll and Schreck, in El Paso, Texas. Dr. T. I. Means, of Santa Fe, assisted Drs. Koll and Schreck.

The afternoon was spent in a tour of the veterinary hospital at Fort Bliss, conducted by members of the staff.

Resolutions were adopted thanking the American Veterinary Medical Association for making it possible for President Foster to attend the meeting, and approving the plan suggested by President Foster for publishing the proceedings of each annual meeting in one volume, provided that it did not necessitate increasing the amount of the annual dues unreasonably.

TRUMAN I. MEANS, *Secretary.*

NEW HAMPSHIRE VETERINARY MEDICAL ASSOCIATION

The annual meeting of the New Hampshire Veterinary Medical Association was held at the State House, Concord, January 5, 1937.

The program consisted of the following subjects:

"Bang's Disease Testing and Tuberculin Testing," by Dr. E. A. Crossman, B. A. I. Inspector-in-Charge, Boston, Mass., and Dr. R. W. Smith, State Veterinarian of New Hampshire, Concord.

The proper methods of preparing expense accounts pertaining to Federal testing for Bang's disease.

"An Outbreak of Swamp Fever in New Hampshire," by Dr. F. F. Russell, of Concord.

The election of officers resulted as follows: President, Dr. Dana H. Lee, Lancaster; vice-president, Dr. Carl E. Chase, Manchester, and secretary-treasurer, Dr. C. L. Martin, University of New Hampshire, Durham. A motion was passed to give the power to the new president to appoint the A. V. M. A. delegate to the House of Representatives.

C. L. MARTIN, *Secretary.*

Observation

When cows are fed on fodder,
One end moos but not the udder.

—*Lantern.*

NECROLOGY

ORVAL HERBERT HOSTETLER

Dr. Orval H. Hostetler died in the Samaritan Hospital, Nampa, Idaho, August 10, 1936, following three operations for gallstones during a period of nine weeks.

Born in McPherson, Kan., June 5, 1903, Dr. Hostetler attended grade and high schools in Nampa, Idaho, and then spent three years in the College of Idaho. His veterinary training was received at the State College of Washington. He was graduated in 1930. Prior to his illness, he was associated in practice with his father, Dr. F. H. Hostetler, who has practiced in Nampa for 30 years.

Dr. Hostetler joined the A. V. M. A. in 1930. He was a member of Alpha Psi fraternity.

J. VON H. SCHANTZ

Dr. J. Von H. Schantz, of Fairfield, Iowa, died December 6, 1936, following an operation for appendicitis. He was a graduate of Iowa State College, class of 1917, and enjoyed a very extensive practice at Fairfield, where he had lived most of the time since his graduation. At the time of his death, Dr. Schantz was engaged in testing for Bang's disease on a per diem basis, and previously he had participated in coöperative tuberculosis eradication work.

J. A. B.

KIRBY D. WERLEY

Dr. Kirby D. Werley, of Hamburg, Pa., died in the Haff Hospital, Northampton, Pa., December 20, 1936.

Born in Albany Township, Berks County, Pa., June 4, 1886, Dr. Werley attended local schools and then entered Keystone State Normal School (now Kutztown State Teachers College). Following his graduation, he decided to study veterinary medi-

cine and entered McKillip Veterinary College, Chicago. He was graduated in 1908 at the head of his class. He returned to Hamburg and entered general practice. Gradually he got into live stock raising and farming, until his holdings exceeded 400 acres and a large herd of cattle.

Dr. Werley was active in civic affairs and was affiliated with a number of fraternal organizations. He is survived by his widow (née Fannie Moyer), four sons and three daughters.

M. D. R.

CHARLES VICTOR NOBACK

Dr. Charles V. Noback, of Scarsdale, N. Y., veterinarian to the New York Zoölogical Park, the Bronx, for the past ten years, died in the Columbia-Presbyterian Medical Center, January 16, 1937, after a brief illness that resulted from a severe cold contracted about a week before his death. He had attended the veterinary conference at Cornell University, January 7-8.

Born in New York City, March 2, 1888, Dr. Noback received his veterinary training at Cornell University. Following his graduation in 1911, he served as a veterinary inspector in the U. S. Bureau of Animal Industry for two years. He then accepted a position with the New York City Department of Health and continued in this work until 1920, when he resigned to accept a position in the Laboratory of Hygiene, Bogota, Colombia. Here he was pathologist and consulting veterinarian on tropical animal diseases, at the same time teaching in the Veterinary School of the National University of Colombia.

In 1925, Dr. Noback returned to the United States to accept a position as bacteriologist with the New York Department of Health, at Albany. The same year he received his Ph. D. from Cornell University. In the latter part of 1926, he joined the staff of the New York Zoölogical Park. During the ten years that he was engaged in this work, he made a number of valuable contributions to the literature on diseases of wild animals.

Dr. Noback joined the A. V. M. A. in 1913. He was a member of the New York State Veterinary Society and was president of the Veterinary Medical Association of New York City at the time of his death. He was also president of the Alumni Association of the New York State Veterinary College at Cornell University. He was a fellow of the New York Zoölogical Society, of the New York Academy of Sciences and of the American Association for the Advancement of Science. Dr. Noback's other affiliations included the American Association of Physical Anthropologists and

the American Society of Mammalogists. He was a member of the Twelfth International Veterinary Congress and took a prominent part as a member of the Committee on Local Arrangements for the New York Congress. He held a commission as captain in the Veterinary Reserve Corps. Surviving Dr. Noback are his widow, two sons, a daughter, a sister and a brother.

EDGAR C. HOWELL

Dr. Edgar C. Howell, of La Mesa, Calif., died in the Naval Hospital at San Diego, Calif., January 17, 1937, at the age of 50 years, after a long illness. He was seriously injured in an automobile accident in July, 1934.

A native of Pennsylvania, Dr. Howell attended Swarthmore (Pa.) Preparatory School and West Chester (Pa.) State Normal College before entering the University of Pennsylvania for the study of veterinary medicine. Following his graduation in 1912, he entered practice at Bishop, Calif. Subsequently he was located in Nevada and Oregon for brief periods.

Dr. Howell joined the A. V. M. A. in 1916. He was a member of Epsilon Chapter, Alpha Psi Fraternity. He is survived by his widow, two sons and three daughters.

Our sympathy goes out to Dr. Gustave A. Kay, of Cedar Rapids, Iowa, in the death of his wife, Clara B. Kay, on December 7, 1936, following an attack of heart trouble, and to Dr. J. W. Connaway, of Columbia, Mo., in the death of his wife, Nellie Ficklin Connaway, on December 17, 1936, at the age of 71 years.

PERSONALS

Dr. H. A. Seidell (Iowa '17), of Des Moines, Iowa, has been reappointed chief of the Iowa Division of Animal Industry.

Dr. J. Earl Gilfillan (U. P. '13), of Galena, Ind., was kicked by a horse recently and was confined to his home for several days.

Dr. Carl W. Groppe (O. S. U. '32), of Wheeling, W. Va., was reappointed Ohio County Veterinarian, on January 1.

Dr. R. W. Grossman (McK. '13), of Columbia, Ill., has been appointed Saint Clair County Veterinarian by the Board of Supervisors.

Dr. J. I. Neal (K. C. V. C. '15), of Southern Pines, N. Car., has resumed practice after a long rest in the interests of his health.

Dr. Jesse C. Brock (K. S. C. '35), of Sale City, Ga., is now associated with Dr. L. J. Herring (K. C. V. C. '09), at Wilson, N. Car.

Dr. A. J. Osteen (Ga. '28) has removed from Wilson, N. Car., and is now practicing at Williamston, N. Car.

DR. A. L. GEFFERT (Ont. '36), formerly of Reedsburg, Wis., has located in Shullsburg, Wis., for general practice.

DR. BYRON P. MERRICK (O. S. U. '23), of Berlin Heights, Ohio, is adding another unit to his small-animal hospital.

DR. P. E. NULPH (Gr. Rap. '12), of Milltown, Wis., has removed to Campbellsport, Wis., for general practice.

COL. JAMES R. SHAND (Chi. '07), U. S. Army retired, recently completed the construction of a new home at 1156 Forest Ave., Palo Alto, Calif.

DR. C. HARVEY SMITH (Chi. '09), of Crown Point, Ind., has been reappointed Lake County Veterinarian by the Board of County Commissioners.

DR. WALTER L. MARTIN (K. C. V. C. '11), recently of Tulsa, Okla., is now in Saint Louis, Mo., having accepted a position as municipal meat inspector there.

DR. W. D. ODOU (Chi. '17), who was located in Hettinger, N. Dak., until June last, gives his present address as 105 W. Washington Ave., Montebello, Calif.

DR. M. W. BATES (O. S. U. '32), of Columbus, Ohio, was elected president of the Hilltop Business Men's Association at the annual meeting in December.

DR. H. K. BAILEY (Cin. '15), of Wilmington, Ohio, was elected vice-president of the Clinton County Agricultural Society, at the meeting held early in January.

DR. G. A. WILLIAMS (Wash. '36) is assistant to Drs. G. W. Blanche (Iowa '02) and D. A. Adler (Wash. '31), at the Glendale Small Animal Hospital, Glendale, Calif.

DR. J. L. CHERRY (U. S. C. V. S. '13), formerly of Tarkio, Mo., has accepted a position at the Virgin Islands Agricultural Experiment Station, Christiansted, Saint Croix.

DR. J. O. R. CAMPBELL (Chi. '07), of Geneva, Ind., broke both legs when he was thrown from his saddle horse on January 18. He was taken to a hospital in Decatur.

DR. G. W. BUTLER (Ont. '84), of Indianapolis, Ind., was a hospital patient the first 18 days of December. The latest report is to the effect that prospects for recovery are good.

DR. E. L. SIDWELL (K. C. V. C. '15), of Hardin, Ill., was seriously injured in an automobile accident, near Palmyra, Ill., on January 12. He was taken to a hospital in Carlinville.

DR. CHARLES HAASJES (Gr. Rap. '18), of Shelby, Mich., has been "having a great time" at Brooks Camp, Sarasota, Fla., according to a postal card received at the JOURNAL office.

DR. HOWARD L. DEUELL (K. C. V. C. '10), of Peoria, Ill., escaped serious injury on January 2, when his car was hit by another machine and overturned at an intersection in Peoria.

DR. EDGAR WRIGHT (Ont. '21), of Bluffton, Ind., was elected president of the Indiana Ayrshire Association, at the annual meeting held at Purdue University during the agricultural conference in January.

DR. B. F. CORBIN (O. S. U. '35), who has been practicing in Venice, Calif., has taken over the Los Angeles Dog and Cat Hospital, in Hollywood, formerly conducted by Dr. Joseph J. Zilligen (U. P. '06).

DR. J. G. HARDENBERGH (U. P. '16), of the Walker-Gordon Laboratory Company, Plainsboro, N. J., gave a talk on "Control Methods in the Production of Certified Milk," before the Students' Dairy Science Association of the New Jersey College of Agriculture, on November 23, 1936.